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SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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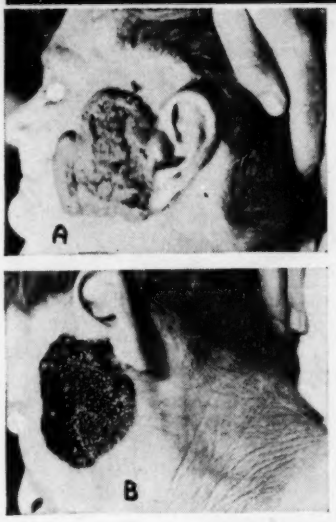

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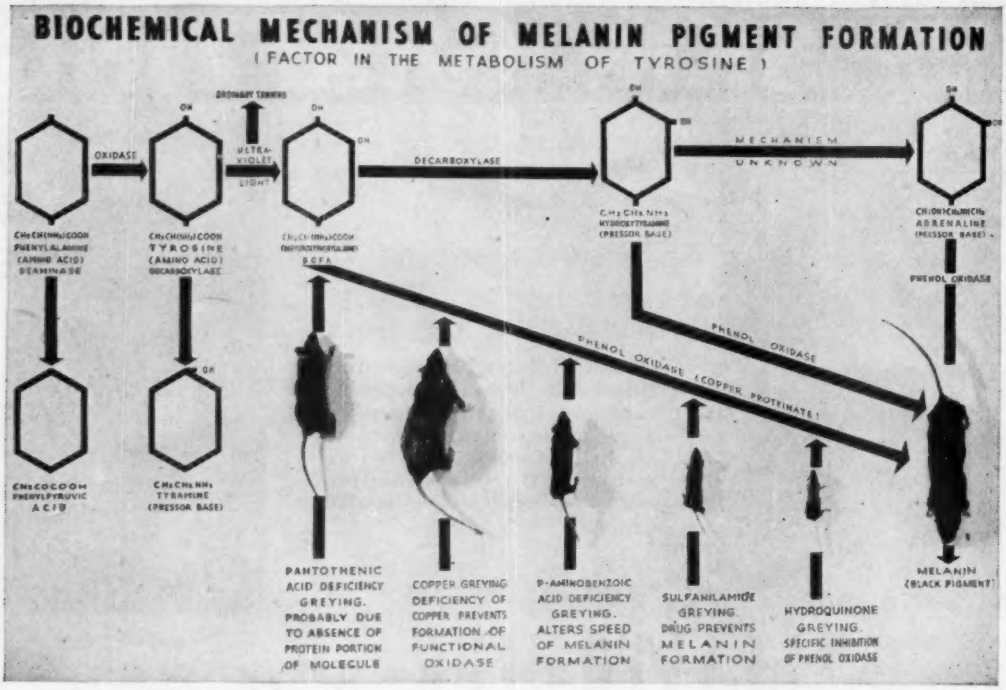
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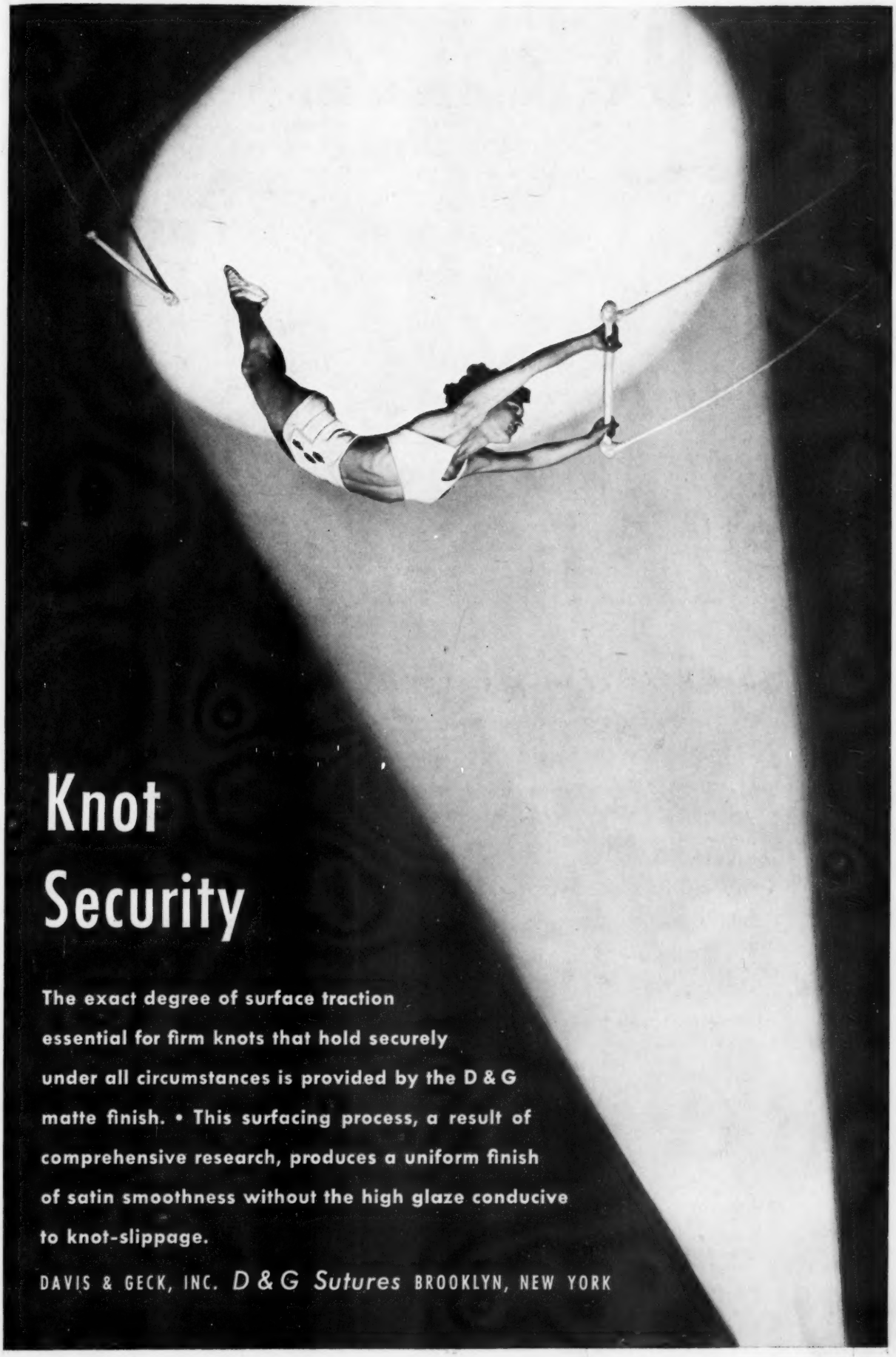

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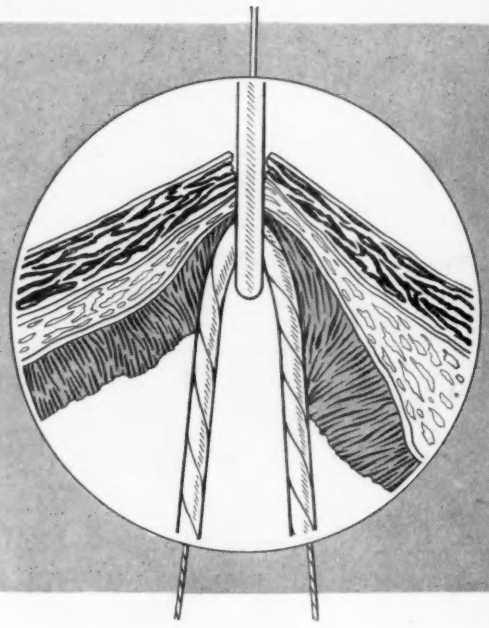

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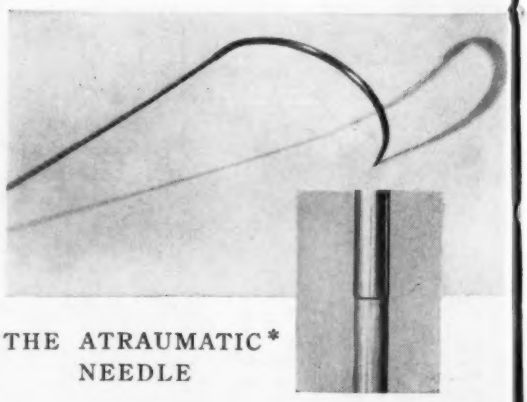

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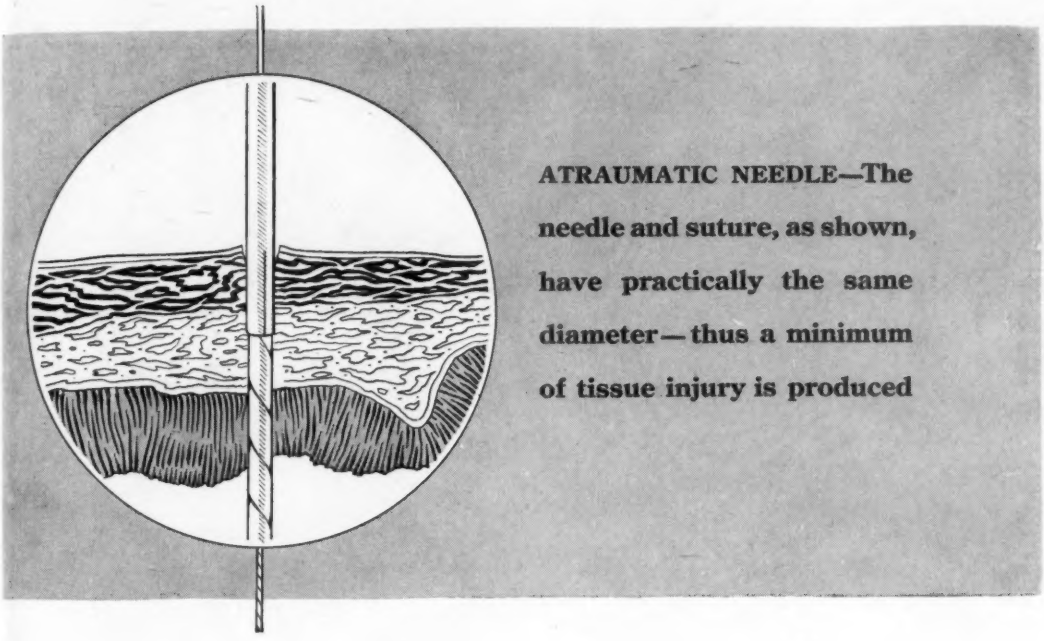

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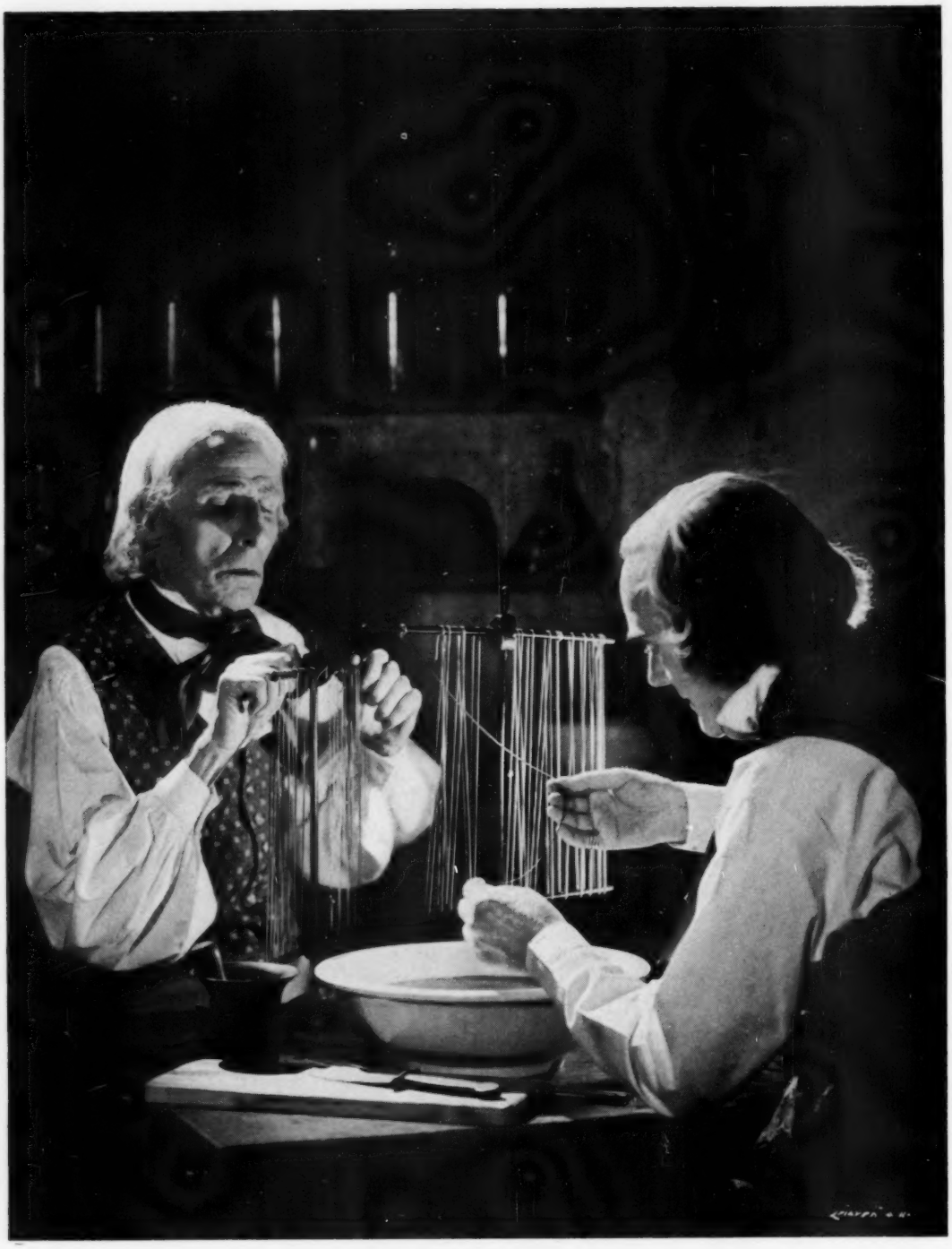

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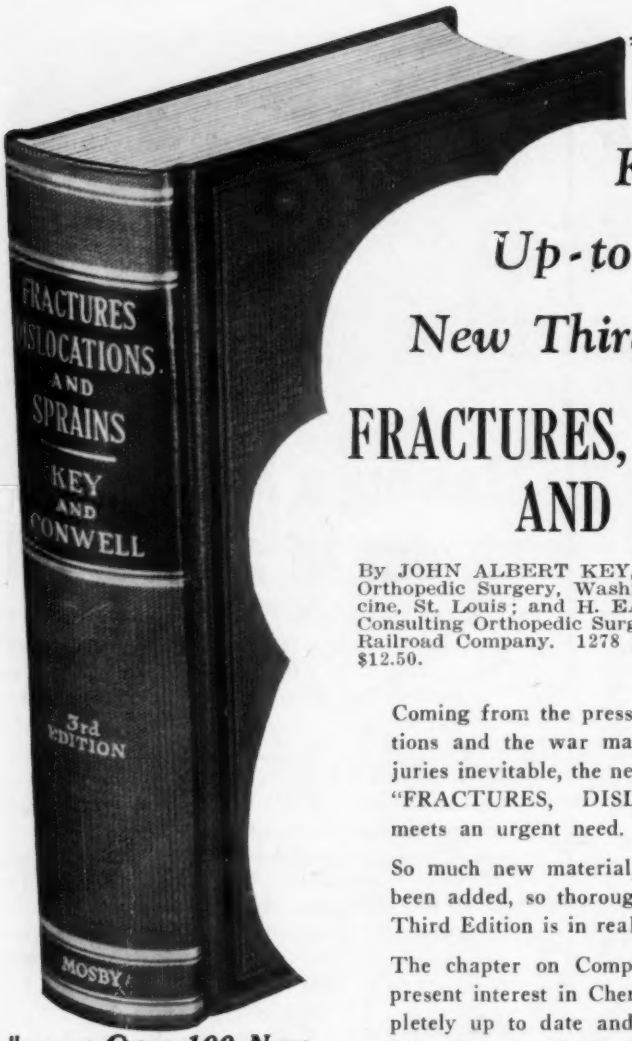

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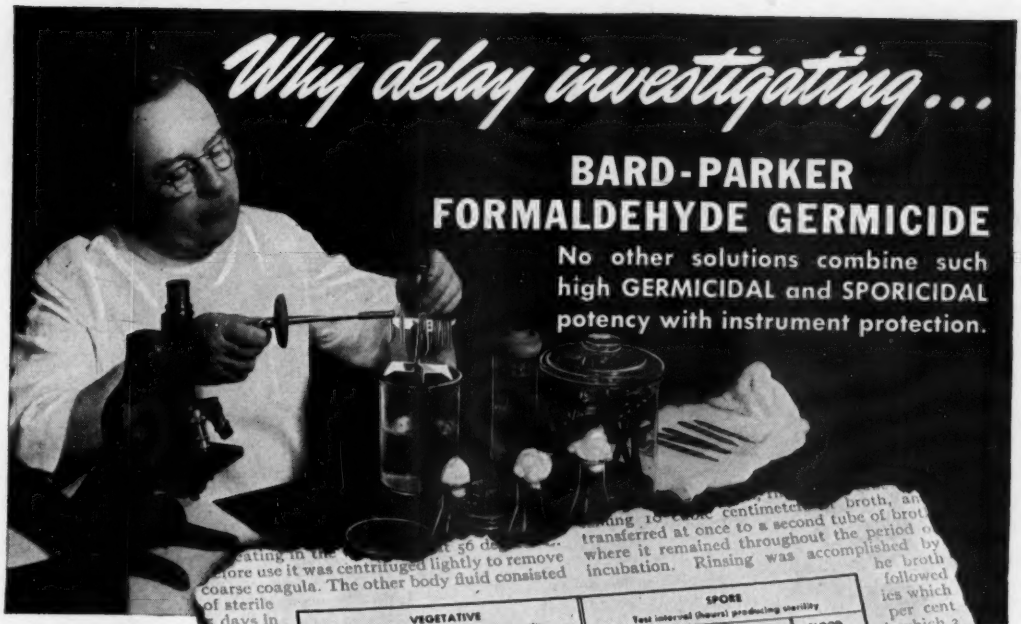
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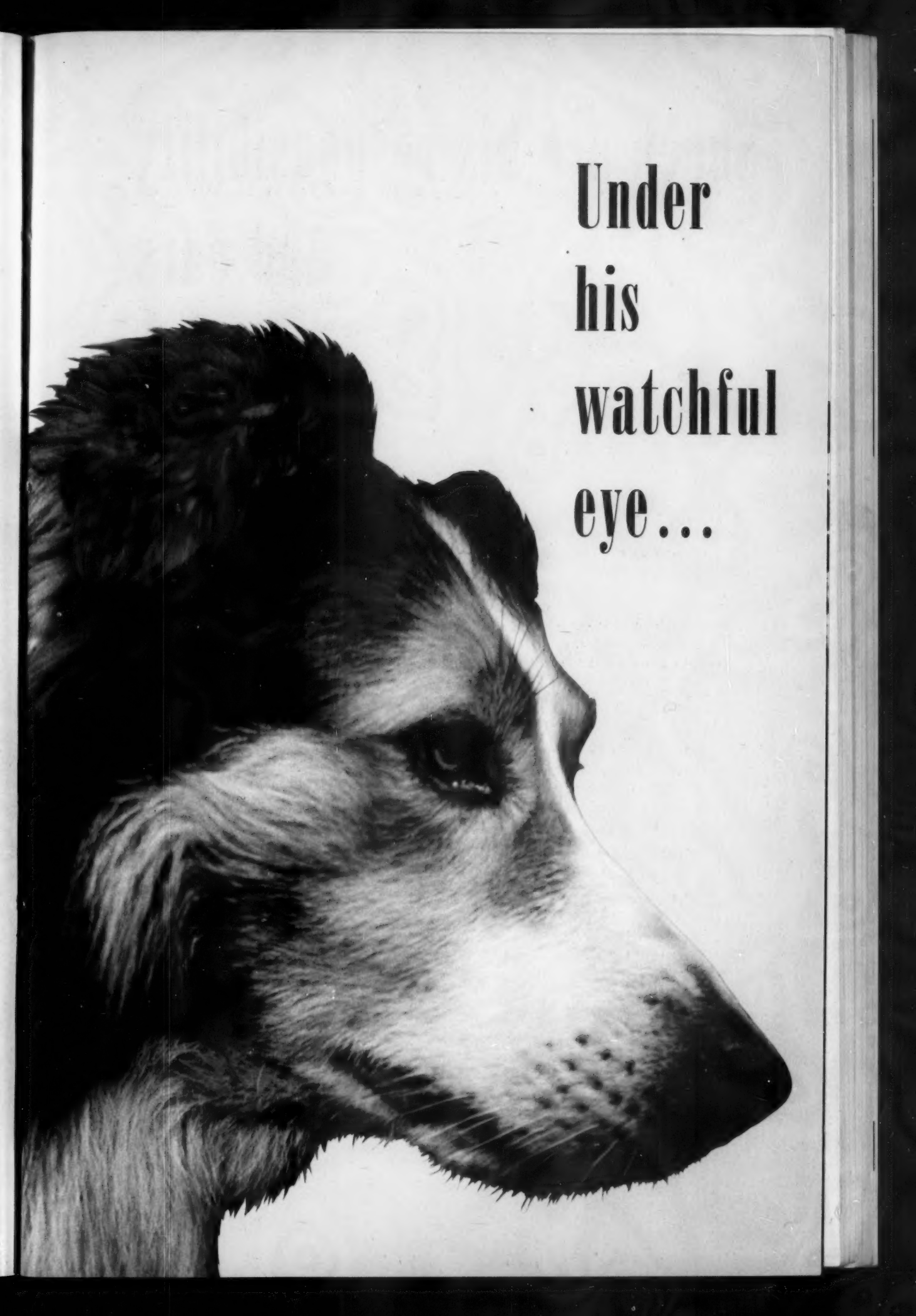
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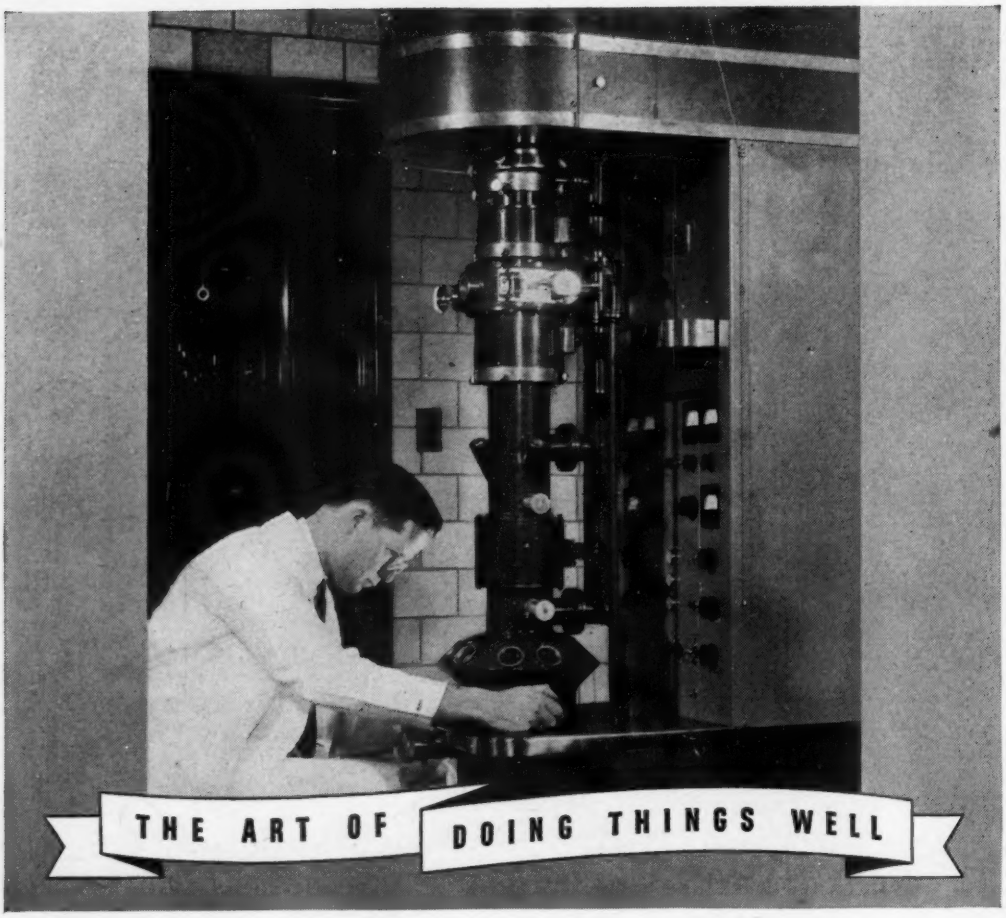

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SURGERY

VOL. 12 JULY, 1942 NO. 1

Original Communications

THE EFFECT OF ADRENAL CORTICAL HORMONES IN HEMORRHAGE AND SHOCK*

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IN A NUMBER of recent publications[1-4] a favorable influence on secondary shock has been claimed for the adrenal cortical hormones. In our view the validity of such claims can be judged best in terms of the ability of these hormones to correct a quantitative deficiency in the volume of circulating plasma, as this is the most characteristic objective feature of shock. Although measurements of plasma volume may not be valid during the actual state of shock, information of value in this respect is obtainable during hemorrhage before the onset of shock and while plasma volume determinations are still reliable. The purpose of this communication is to report our observations on the effect of the adrenal cortical hormones, first, on the circulating plasma volume in hemorrhage before the onset of shock, and second, on the blood pressure curve and survival time following the onset of shock due to hemorrhage.

THE INFLUENCE OF ADRENAL CORTICAL HORMONES ON THE CIRCULATING PLASMA VOLUME AND PLASMA PROTEIN IN HEMORRHAGE

1. *Massive Hemorrhage Without Producing Shock.*—The volume of circulating plasma and the total plasma protein of normal unanesthetized dogs, deprived of food, but not water, for the preceding twenty-four hours was determined by the Evans' blue dye and specific gravity methods respectively.[5, 6] They then were bled over a period of ten to twenty minutes (by needle puncture of the jugular vein or the femoral artery) of some 30 to 40 per cent of their estimated† blood volume.

*The expenses of this investigation were defrayed in part by the Proctor Fund, Harvard University.

†The figures for the original circulating plasma volume were not available until some time after the bleeding was performed.

Received for publication, Oct. 18, 1941.

Shock did not result. The dogs remained active and frisked about, apparently unharmed by the massive hemorrhage. Four hours later the circulating plasma volume and total plasma protein were again determined. From these data the compensatory mobilization of fluid and protein from extravascular depots was then calculated.

Such observations were made in eight different groups of dogs as shown in the accompanying tables (Tables I and II).

A. *The Mobilization of Fluid:* Five control dogs (Group I) regained an average of 53 per cent of the plasma volume lost. Eight dogs (Group II) in which cortin* was injected intravenously immediately after the hemorrhage, in doses varying from 2 c.c. to 10 c.c. (100-500 dog units), regained an average of 67 per cent of the plasma volume lost. Four dogs (Group III) receiving 150 c.c. of physiologic saline solution distributed widely in the subcutaneous tissues immediately after the hemorrhage, regained an average of 79 per cent of the plasma volume lost. Seven dogs (Group IV) were given cortin, as in Group II, together with saline solution, as in Group III. The return of lost plasma volume to the circulation averaged 90 per cent. Four dogs (Group V) were given 150 c.c. of plasma subcutaneously, distributed widely as was the saline solution in Group III. The compensatory plasma return averaged 68 per cent. In five dogs (Group VI) which were given cortin, as in Group II, and plasma, as in Group V, the compensatory plasma return averaged 81 per cent.

From an analysis of the data in Table I† it appears that cortin does not effect a better mobilization of extravascular fluid than will occur without it. The presence of an interstitial reservoir of saline solution by itself will achieve as much. However, when cortin and saline solution are provided together, the advantage is substantial in that almost all of the lost plasma volume is replaced within the four-hour period.

Following massive hemorrhage which does not result in shock, a certain usefulness therefore may be claimed for cortin if physiologic saline solution is provided subcutaneously.

Three dogs (Group VII) which were given 10 to 15 mg. of desoxycorticosterone acetate‡ intravenously, instead of cortin, mobilized an average of 69 per cent of the plasma volume withdrawn. Five dogs (Group VIII) which received the desoxycorticosterone acetate and saline solution, as in Group III, mobilized an average of 75 per cent, which was about the same as when saline solution alone was given.

From the data in Table I it appears that desoxycorticosterone acetate exerts no significant effect on the mobilization of fluid into the blood

*Kindly furnished by The Upjohn Company as Solution of Adrenal Cortical Extract.

†The conclusions drawn in the subsequent text as to the role of the hormone in the mobilization of fluid and of protein are based on a statistical analysis of the data. Positive results are claimed only for the data in Groups IV and VI of Table I and Group VI in Table II. "P" equals 4.3, 3.1, and 2.75, respectively, in these three groups.

‡Kindly furnished by Ciba Pharmaceutical Products, Inc., under the trade name Percorten.

stream after hemorrhage which does not result in shock, whether or not an interstitial reservoir of saline solution is provided.

Three of the five control dogs (Group I) died within twenty-four hours, whereas all other animals, except one in Group IV which had been bled on a previous occasion, survived. This does not prove that the hormones possess any special virtue under the conditions of these experiments because those dogs receiving saline solution alone or plasma alone also survived.

COMMENT. One of the difficulties in a comparative evaluation of the results of different investigators arises from marked differences in the dosages of the hormones utilized. For example, Weil, Rose, and Browne[3] gave rats 10 c.c. of cortin plus 10 mg. of desoxycorticosterone acetate before and 10 c.c. of cortin plus 10 mg. of desoxycorticosterone acetate after trauma. Our doses, which in proportion to body weight were only a small fraction of these, approximate effective physiologic dosages much more closely and are, therefore, more consistent with practical therapeusis. There was, however, no noticeable difference in the effect between the smallest and largest dosages which we utilized.

B. *The Mobilization of Protein:* Table II shows the average per cent of the circulating plasma protein lost by hemorrhage which was regained in the subsequent period of four hours, as follows: Group I (control), 30 per cent; Group II (cortin), 30 per cent; Group III (saline solution), 27 per cent; Group IV (cortin plus saline solution), 46 per cent; Group V (plasma), 32 per cent; Group VI (cortin plus plasma), 62 per cent; Group VII (desoxycorticosterone acetate), 24 per cent, and Group VIII (desoxycorticosterone plus saline solution), 31 per cent.

Cortin alone, therefore, seems unable to produce any favorable effect on the compensatory mobilization of lost plasma proteins. Saline solution alone likewise offers no advantage in this respect. The value of cortin plus saline solution is doubtful. But cortin plus plasma achieves an appreciable result. Desoxycorticosterone acetate is wholly ineffective, whether given alone, with saline solution or, as in one unlisted experiment, with plasma.

COMMENT. The mobilization of protein by cortin is presumably dependent, in some degree at least, on the amount of protein available in reserve depots, because the cortin was inactive until an extra supply of plasma was provided. An extra supply of saline solution or of plasma alone in the four-hour period of observation did not succeed in adding protein to the circulation.

The ratio between the amount of protein mobilized and the volume of plasma fluid mobilized varied widely in all groups (except Groups IV and VI, in which the individual ratios showed less disparity). Perhaps this is an indication of a considerable variation in the amount of plasma protein in reserve, which is to be expected among laboratory animals whose nutritional status varies widely.

TABLE I

EFFECT OF ADRENAL CORTICAL HORMONES ON PLASMA VOLUME RECOVERY FOUR HOURS AFTER MASSIVE HEMORRHAGE IN NORMAL UNANESTHETIZED DOGS

GROUP NO.	HORMONE (WITH OR WITHOUT PARENTERAL FLUID)	DOSE OF HORMONE	ORIGINAL PLASMA VOLUME WITHDRAWN (%)	LOST PLASMA VOLUME MOBILIZED (%)
I	No hormone; no parenteral fluid	-	33	52
		-	43	26
		-	37	67
		-	44	53
		-	31	65
			Average	53
II	Cortin; no parenteral fluid	2 c.c.	35	88
		2 c.c.	40	44
		2 c.c.	41	66
		2 c.c.	39	55
		2 c.c.	26	43
		2 c.c.	38	77
		4 c.c.	35	96
		10 c.c.	36	63
			Average	67
III	No hormone; subcutaneous saline solution	-	24	72
		-	28	120
		-	41	54
		-	33	71
			Average	79
IV	Cortin and subcutaneous saline solution	2 c.c.	36	118
		2 c.c.	40	114
		2 c.c.	46	80
		10 c.c.	38	83
		10 c.c.	43	84
		10 c.c.	36	74
		10 c.c.	40	77
			Average	90
V	No hormone; subcutaneous plasma	-	32	32
		-	35	80
		-	40	72
		-	39	87
			Average	68
VI	Cortin and subcutaneous plasma	2 c.c.	44	126
		2 c.c.	39	49
		10 c.c.	42	91
		10 c.c.	26	45
		10 c.c.	37	96
			Average	81
VII	Desoxycorticosterone acetate; no parenteral fluid	10 mg.	35	76
		15 mg.	37	54
		15 mg.	31	76
			Average	69
VIII	Desoxycorticosterone acetate; subcutaneous saline solution	10 mg.	34	81
		10 mg.	35	69
		10 mg.	38	78
		15 mg.	30	45
		15 mg.	35	100
			Average	75

TABLE II

EFFECT OF ADRENAL CORTICAL HORMONES ON PLASMA PROTEIN RECOVERY FOUR HOURS AFTER MASSIVE HEMORRHAGE IN NORMAL UNANESTHETIZED DOGS

GROUP NO.	HORMONE (WITH OR WITHOUT PARENTERAL FLUID)	DOSE OF HORMONE	ORIGINAL PLASMA PROTEIN WITHDRAWN (%)	LOST PLASMA PROTEIN MOBILIZED (%)
I	No hormone; no parenteral fluid	-	33	27
		-	43	26
		-	37	16
		-	44	34
		-	31	51
			Average	30
II	Cortin; no parenteral fluid	2 c.c.	35	38
		2 c.c.	40	22
		2 c.c.	41	36
		2 c.c.	39	32
		2 c.c.	26	1.7
		2 c.c.	38	39
		4 c.c.	35	50
		10 c.c.	36	21
			Average	30
III	No hormone; subcutaneous saline solution	-	24	0
		-	28	69
		-	41	21
		-	33	19
			Average	27
IV	Cortin and subcutaneous saline solution	2 c.c.	36	76
		2 c.c.	40	48
		2 c.c.	46	40
		10 c.c.	38	46
		10 c.c.	43	50
		10 c.c.	36	30
		10 c.c.	40	21
			Average	46
V	No hormone; subcutaneous plasma	-	32	5
		-	35	36
		-	40	32
		-	39	55
			Average	32
VI	Cortin and subcutaneous plasma	2 c.c.	44	100
		2 c.c.	39	27
		10 c.c.	42	67
		10 c.c.	26	48
		10 c.c.	37	69
			Average	62
VII	Desoxycorticosterone acetate; no parenteral fluid	10 mg.	35	29
		15 mg.	37	15
		15 mg.	31	28
			Average	24
VIII	Desoxycorticosterone acetate; subcutaneous saline	10 mg.	34	30
		10 mg.	35	19
		10 mg.	38	50
		15 mg.	30	14
		15 mg.	35	41
			Average	31

2. *Massive Hemorrhage Producing Shock.*—If the hormones are to be regarded as effective therapeutic agents in shock, their beneficial action should be demonstrable when hemorrhage or any other trauma has resulted in a state of shock, for in this state there is a far greater

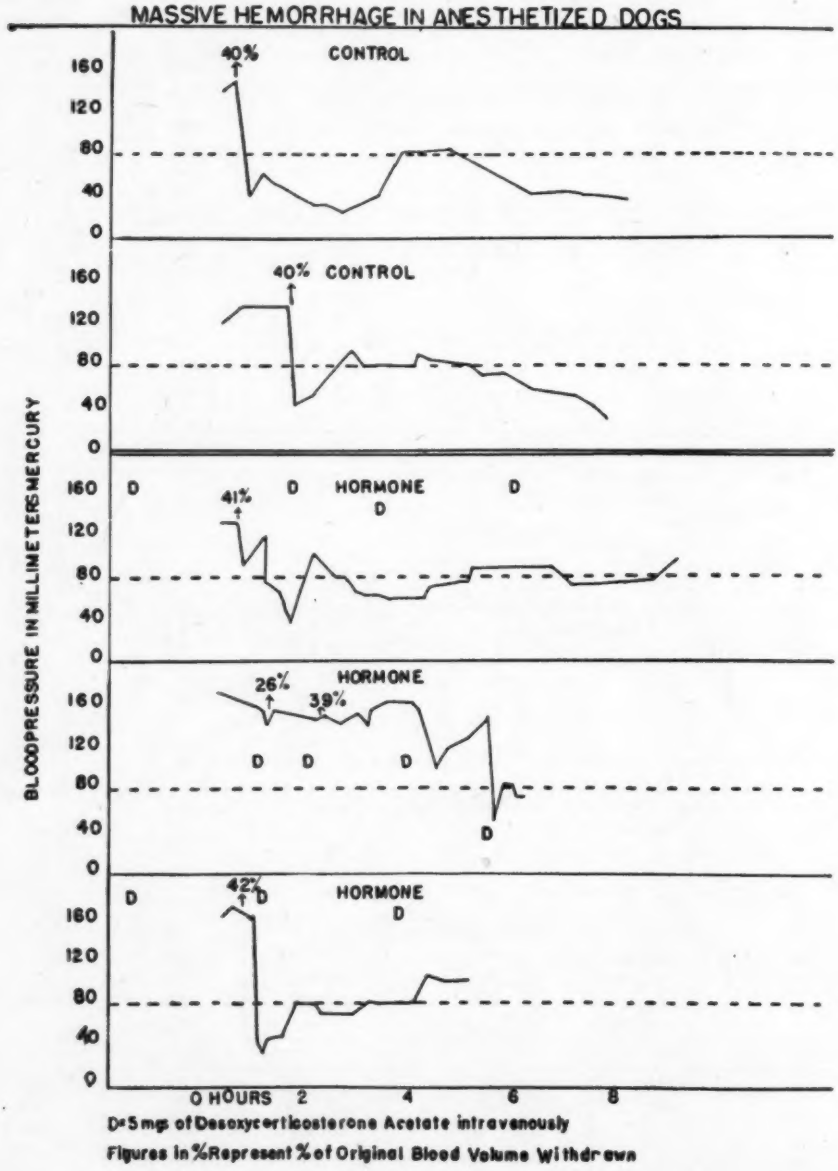

Fig. 1.

need for compensation than is the case under the conditions described in the preceding experiments. But plasma volume determinations in the shock state may not be reliable, so that a quantitative estimate of the capacity of the hormones to mobilize extravascular fluids during shock is perhaps not possible. A favorable response to the hormones

during shock can at present be assumed only if they effect a sustained return of the blood pressure level to well above the shock level and (or) a significant prolongation of the survival time.

Observations along these lines were made as follows: A group of dogs was submitted to massive hemorrhage. Heparinization and intraperitoneal nembutal anesthesia were utilized in order to record continually the blood pressure in the carotid artery. In contrast to unanesthetized dogs which survived an acute blood volume loss of some 40 per cent without going into shock, the anesthetized dogs uniformly and promptly went into shock. A systolic blood pressure level of 80 mm. Hg or lower for thirty minutes or longer was regarded as evidence of a state of shock.

Two dogs so prepared were rapidly bled of 40 per cent of their estimated blood volume. Their blood pressure curves promptly fell to shock level and death occurred in about seven hours (Fig. 1).

Three dogs treated in similar fashion were given five mg. of desoxycorticosterone acetate intravenously at intervals before and during shock as indicated in Fig. 1. This hormone exerted a slight but insignificant improvement on the course of events. Similarly, cortin given to two such dogs produced no substantial benefit.

That we were not dealing with a type of shock from which recovery was impossible and which, therefore, may have masked the possible sustaining powers of the hormones is evident in the case of five dogs, all of which recovered after being in profound shock for one-half hour or longer, following an intravenous injection of 500 c.c. of saline solution or 200 c.c. of plasma (Fig. 4 and Table III).

TABLE III

THE EFFECT OF INTRAVENOUS FLUIDS WITH AND WITHOUT CORTIN ON THE TIME NEEDED FOR RECOVERY FROM A CONSTANT SHOCK LEVEL TO A STABILIZED RECOVERY LEVEL OF BLOOD PRESSURE

EXPERIMENT NO.	INTRAVENOUS TREATMENT APPLIED 30 OR MORE MINUTES AFTER ONSET OF SHOCK	B.P. BEFORE HEMORRHAGE (MM. HG)	ORIGINAL BLOOD VOLUME WITHDRAWN (%)	STABILIZED B.P. AFTER TREATMENT (MM. HG)	TIME NEEDED TO REACH STABILIZED RECOVERY LEVEL OF B.P. FOLLOWING PERSISTENT SHOCK LEVEL OF B.P.
1	500 c.c. saline solution intravenously	130	35	80	120 min.*
2		150	43	130	145 min.
3		155	55	118	60 min.
4	10 c.c. cortin; 500 c.c. saline solution intravenously	185	56	140	20 min.
5		140	35	100	Almost immediately
6		150	44	115	Almost immediately
7		140	50	110	Almost immediately
8		170	41	145	Almost immediately
9	200 c.c. plasma intravenously	160	45	125	185 min.
10		150	31	90	120 min.
11	10 c.c. cortin; 200 c.c. plasma intravenously	110	40	105	95 min.
12		145	18	125	216 min.

*Recovery level did not rise above 80 mm.; dog died 48 hours later.

Although cortin is without effect during shock in the absence of intravenous saline solution or plasma, it apparently possesses a slight adjuvant quality if combined with saline solution. The return of the blood pressure to a sustained recovery level occurred almost *immediately* in four out of five dogs which received 10 c.c. of cortin in addition to 500 c.c. of physiologic saline solution intravenously, in contrast to a recovery after 145 minutes, 120 minutes, and 60 minutes, respectively,

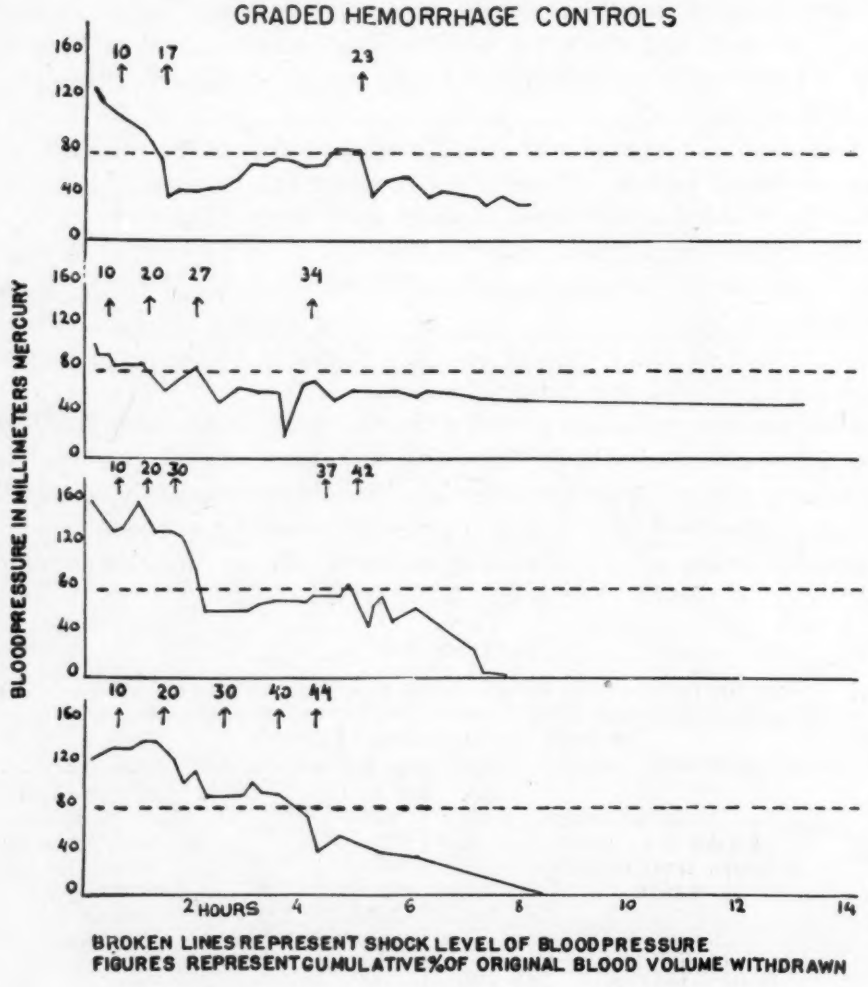

Fig. 2.

in three dogs receiving saline solution only. Plasma alone restored the blood pressure in two dogs after 185 and 120 minutes, respectively, while 10 c.c. of cortin plus plasma effected the same result after 216 and 95 minutes, respectively (Fig. 4 and Table III).

3. *Graded Hemorrhage Producing Shock.*—Since a single massive hemorrhage precipitates shock in the anesthetized dog almost immediately, it is possible that too little time is available for the organism to

benefit by whatever fluid mobilizing capacity the hormones might exert under less strenuous circumstances. Accordingly, the above experiments were repeated, substituting for a single massive hemorrhage, successive withdrawals every forty to sixty minutes, of an estimated 10 per cent of the original total blood volume so as to prolong the period preceding the onset of shock and to allow more time for the mobilization of extravascular fluids.

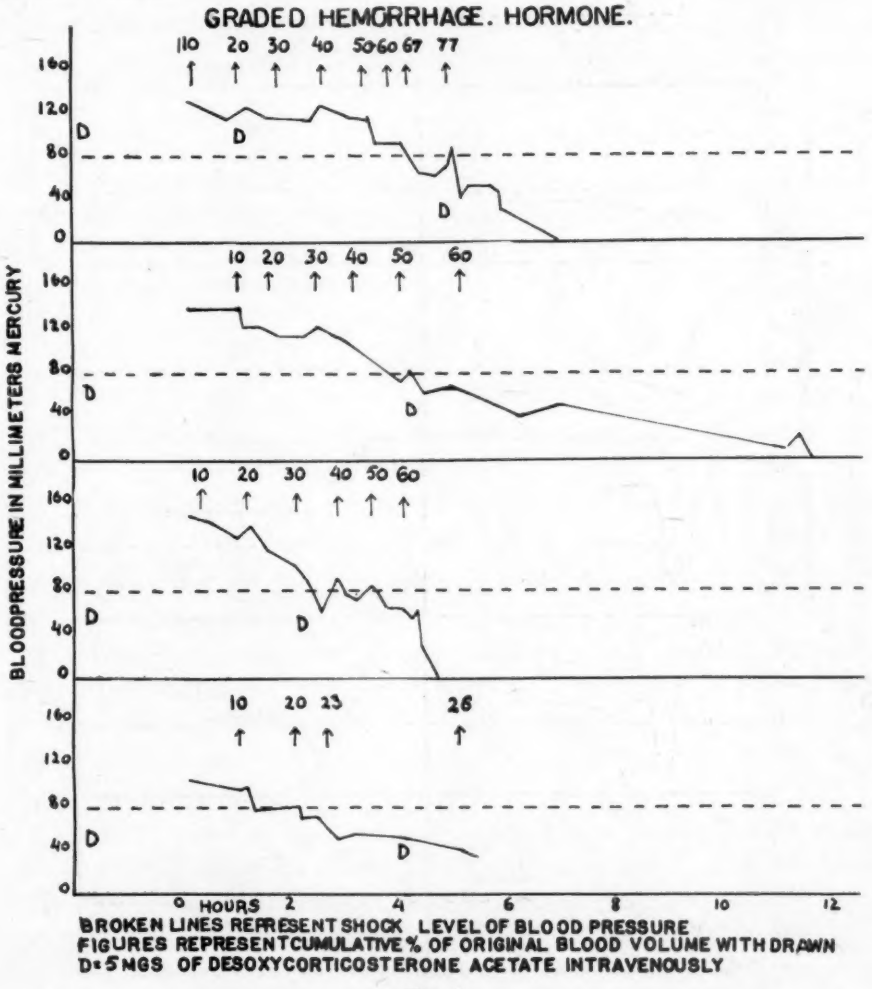
GRADED HEMORRHAGE. HORMONE.
BLOODPRESSURE IN MILLIMETERS MERCURY
HOURS
BROKEN LINES REPRESENT SHOCK LEVEL OF BLOOD PRESSURE
FIGURES REPRESENT CUMULATIVE % OF ORIGINAL BLOOD VOLUME WITH DRAWN
D=5 MGS OF DESOXYCORTICOSTERONE ACETATE INTRAVENOUSLY

Fig. 3.

In preliminary experiments it was soon apparent that anesthetized dogs vary considerably in resistance to equal degrees of blood loss (Figs. 2 and 3) in so far as the development of shock is concerned. A shock level of blood pressure was reached within two to five hours and death occurred within eight hours in hormone-treated as well as in untreated animals. Nevertheless, in three out of four preliminary experiments with desoxycorticosterone acetate it was necessary to withdraw more blood to induce shock than in the control series (Fig. 3).

Plasma volume determinations were made as often as circumstances allowed on a subsequent series of twelve dogs during the period preceding the onset of shock. Three dogs received desoxycorticosterone

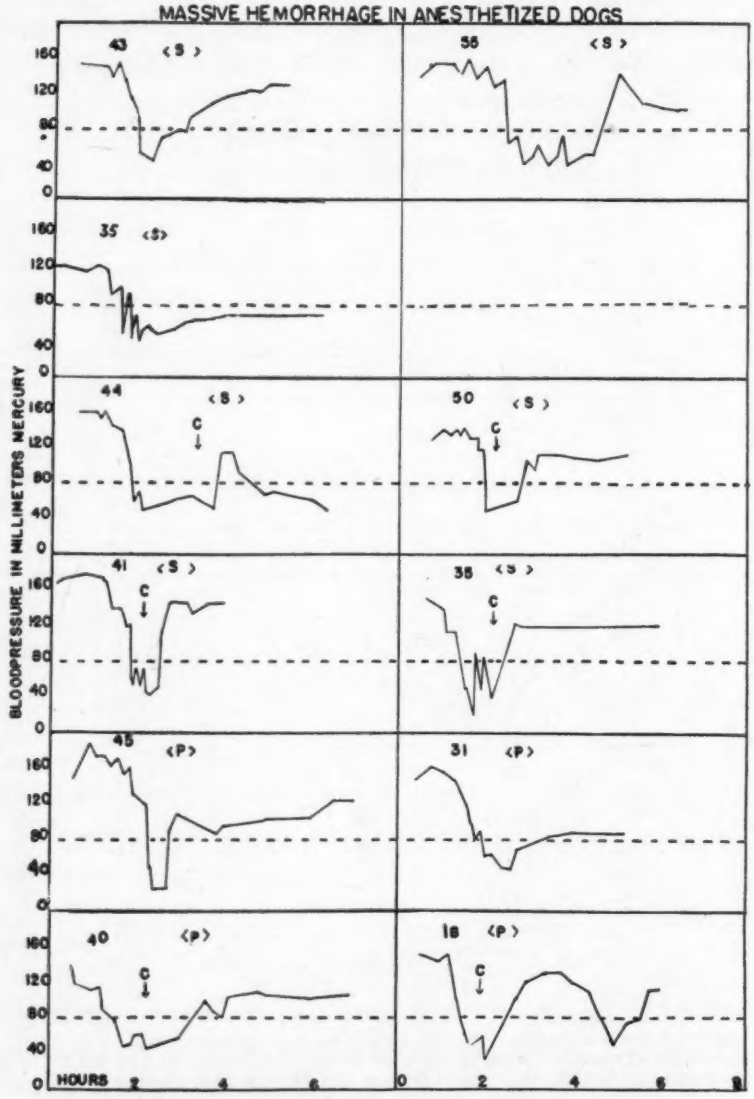
MASSIVE HEMORRHAGE IN ANESTHETIZED DOGS
BLOODPRESSURE IN MILLIMETERS MERCURY
43 (S)
55 (S)
35 (S)
44 (S)
50 (S)
41 (S)
38 (S)
45 (P)
31 (P)
40 (P)
18 (P)
C
HOURS
BROKEN LINES REPRESENT SHOCK LEVEL OF BLOOD PRESSURE
HORIZONTAL ARROWS SHOW TIME DURING WHICH SALINE OR PLASMA WAS GIVEN
VERTICAL ARROW SHOWS TIME OF INJECTION OF CORTIN
S = SALINE (500cc) P = PLASMA (200cc) C = CORTIN (10cc)
FIGURES REPRESENT % OF ORIGINAL BLOOD VOLUME WITHDRAWN

Fig. 4.

acetate and two others received cortin. The hormone was given intravenously two hours before the bleeding began. The remainder received no hormone. Even though it was not possible to obtain plasma volume determinations at corresponding intervals or after equal degrees of blood

loss, data of some value were secured. An inspection of Table IV shows that the hormone-treated dogs have no greater tolerance to graded hemorrhage than control animals, if they have as much. This is evident from (1) the amount of plasma removed in order to induce an irrecoverable shock level of blood pressure, (2) the fluid volume compensation for a given volume of plasma loss, (3) the net loss of plasma volume at the time of onset of the shock level of pressure, (4) the extent of the compensatory mobilization of plasma proteins, and (5) the absence in the hormone-treated dogs of a prolongation of the survival time.

The evidence is therefore against the usefulness of desoxycorticosterone acetate and of cortin in anesthetized animals subjected to massive or graded hemorrhage resulting in shock, *unless parenteral fluid is provided.* Given such a supply of fluid, cortin is advantageous while desoxycorticosterone acetate is not.

SUMMARY

1. Following a single massive hemorrhage (about 40 per cent of the total blood volume), anesthetized dogs go into shock. The survival time is not prolonged by cortin or desoxycorticosterone acetate.

2. When graded hemorrhage is carried out in anesthetized dogs, shock develops more slowly. The hormones do not facilitate the mobilization of extravascular fluids or proteins before the onset of shock, nor do they prolong the survival time.

3. Intravenous plasma and saline solution in such shocked animals are effective resuscitating agents. Cortin in addition to saline solution shortens the time required for restoration of the blood pressure to a sustained recovery level. To this extent cortin may be considered to have a slight usefulness in shock.

4. Normal unanesthetized dogs submitted to a single massive hemorrhage (some 40 per cent of the total blood volume) do not go into shock. The fluid-mobilizing capacity of such dogs during a four-hour period following the hemorrhage is not significantly increased by cortin or desoxycorticosterone acetate. Subcutaneous plasma alone or saline solution alone is equally effective. Cortin given with saline solution accomplished the greatest mobilization of all and therefore may be regarded as of some value in the therapy of acute plasma loss. Desoxycorticosterone acetate in these circumstances is ineffective.

5. The mobilization of protein into the circulating plasma after massive hemorrhage in unanesthetized dogs was not significantly improved by the hormones. However, following the injection of plasma into the subcutaneous tissues, the percentage recovery of lost circulating plasma protein was increased by cortin.

TABLE IV

THE EFFECT OF CORTIN AND DESOXYCORTICOSTERONE ACETATE ON THE RECOVERY OF LOST PLASMA VOLUME AND PLASMA PROTEINS DURING GRADED HEMORRHAGE IN ANESTHETIZED DOGS

EXPERIMENT NO.	DOSE OF HORMONE	ORIGINAL PLASMA VOLUME REMOVED AT TIME OF SECOND* PLASMA VOLUME DETERMINATION (%)	% RECOVERY OF % LOSS AT TIME OF SECOND PLASMA VOLUME DETERMINATION		ORIGINAL PLASMA VOLUME REMOVED AT ONSET OF SHOCK (%)	APPROX. NET LOSS OF PLASMA VOLUME AT ONSET OF SHOCK (IN %)	APPROX. NET LOSS OF PLASMA PROTEIN AT ONSET OF SHOCK (IN %)	SURVIVAL TIME IN HOURS
			PLASMA VOLUME	PLASMA PROTEIN				
				NO HORMONE				
1	-	42	16	(-7)	52	40	35	4½
2	-	38	52	(-7)	48	30	20	5
3	-	40	48	32	50	31	21	5¾
4	-	35	42	14	46	26	15	4¼
5	-	23	94	41	44	23	14	2¾
6	-	21	19	(-14)	30	26	16	4
7	-	20	13	0	40	37	17	¾
				DESOXYCORTICOSTERONE ACETATE				
8	9 mg.	26	(-30)	(-33)	29	43	34	6
9	10 mg.	33	39	(-10)	33	26	20	5
10	5 mg.	20	58	54	20	16	8	4
				CORTIN				
11	3 c.c.	22	50	47	22	30	11	3½
12	4 c.c.	37	73	10	46	19	10	5¾

*The first determination was made at the beginning of the experiment to obtain original plasma volume and plasma proteins.

CONCLUSION

Desoxycorticosterone acetate is not an effective agent in the therapy of shock due to hemorrhage. Cortin given alone is not an effective agent in the therapy of shock. If it is combined with parenteral fluids it exerts some beneficial influence in certain circumstances.

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THE THERAPY OF SHOCK IN EXPERIMENTAL ANIMALS WITH PLASMA PROTEIN SOLUTIONS*

I. Concentrated Plasma as a Hemodiluting Agent in Shock

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UNDER normal conditions the blood volume is principally composed of a fairly constant amount of red blood cells and plasma. In traumatic or posthemorrhagic shock there is a decrease in the effective circulating blood volume resulting from the partial loss of one or both of these constituents in varying amounts. Peripheral stagnation of blood in dilated vessels may further accentuate this abnormality.

It is becoming more apparent that generally the volume deficit per se is more important than the deficit of any one constituent, such as the red cells. This reasoning has resulted from the observation that a very copious hemorrhage is not fatal if the circulating volume be maintained with solutions not containing red blood cells. The anemia which results can usually be treated later without jeopardy to the patient when time is not such a valuable factor.

Although many mechanisms in shock are not well understood, the best therapeutic measure appears to be one which encourages the most rapid replacement of the circulating volume. The solutions most efficient in accomplishing this replacement contain plasma proteins, since such proteins tend to be retained within the vessels and, at the same time, are of a physiologic nature. The solutions that have been used[1-4] contain various protein concentrations, such as 15 to 20 Gm. per cent (concentrated), 5 to 7 Gm. per cent (normal), and 3 to 4 Gm. per cent (dilute).

The present communication demonstrates the ability of concentrated plasma to increase the blood volume in severe posthemorrhagic and traumatic shock in dogs. In these experiments (1940 to 1941) a few animals seemed to be harmed by plasma in normal and concentrated form. The apparent cause for these irregularities will be discussed below. Subsequent experiments (1942) dealing mainly with the apparent shifts of body fluid in shock and following therapy with concentrated and dilute serum are the subjects of a separate report.[5]

METHODS

Healthy dogs under nembutal anesthesia were used. The nembutal was given intravenously in doses of one grain for every five pounds of body weight. There were two groups. In the first group the animals

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were bled through a carotid artery cannula. Multiple bleedings over a period averaging three hours was the usual technique, although in a few instances one or two large bleedings were performed. The amount of blood removed ranged between 2.7 per cent and 5.4 per cent of the body weight with an average of 3.8 per cent. In the second group trauma to both lower extremities was the procedure used. Trauma was inflicted with a metal hammer as moderate blows. This procedure was repeated every fifteen to twenty minutes until the blood pressure reached shock levels. The first two trauma periods lasted five minutes and thereafter one- to three-minute periods were used. In this group two dogs were given histamine intravenously in addition to the trauma. In two other animals a tourniquet was applied about the extremities during trauma and later released. The average blood pressure during shock was 58 mm. Hg, ranging between 10 and 85 mm. The hemorrhagic group remained in shock for an average of 2.3 hours and the traumatic ones for an average of 1.5 hours before treatment.

The canine plasma used was obtained from citrated blood by conventional centrifugation in 250 c.c. bottles. During the collection of blood the donor dogs were subjected to intravenous nembutal anesthesia. All of the plasma was first desiccated from the frozen state by the adtevac process.[6] The dry product was dissolved in one-fourth of the original volume and preserved in the frozen state. It was either administered as such or diluted back to normal with distilled water. Most often dog plasma was used but in isolated cases, as indicated in the tables, human plasma was given. The plasma, whether normal or concentrated, was injected rapidly into the femoral vein by means of a syringe, that is, 50 c.c. in one to five minutes' time.

The blood pressure was taken by means of a carotid cannula attached to a mercury manometer. The falling drop technique[7] was used for the specific gravity determinations. The hemoglobin concentration was obtained by the Sanford-Sheard[8] technique using a Leitz-Mass photoelectric colorimeter. The red cells were counted on each occasion in 20 hemocytometer squares. Wintrobe[9] tubes were used in determining the hematocrits.

The blood samples were collected from either the jugular or the femoral vein. Heparin was used as an anticoagulant.

RESULTS

The essential part of the data is given in the tables. The results of the laboratory tests and the level of the mean arterial pressure are recorded in three periods. The first figures represent the control studies, those of the second group were taken during shock and just prior to treatment, and those of the third group were obtained an average of one and one-half hours after treatment, except in the animals that expired sooner, when the results immediately after treatment were recorded.

TABLE I

HEMORRHAGIC GROUP

NO. AND WEIGHT (KG.)	BLEEDING	TREATMENT	BLOOD SPECIFIC GRAVITY	PLASMA SPECIFIC GRAVITY	HB. (GM. %)	R.B.C. (MILLIONS PER C.MM.)	HEMATOCRIT (%)	B.P. (MM. HG)	REMARKS
I 10.0	300 c.c. One-stage	95 c.c. 4 × conc. canine plasma	1.0529 1.0636 1.0554	1.0271 1.0281 1.030		6.58 8.24 6.77	40.4 54.1 34.0	140 45 120	Sacrificed 2 hr. later
II 6.2	250 c.c. Two-stage	90 c.c. 4 × conc. canine plasma	1.0508 1.0503 1.0430	1.0221 1.0261 1.0243		5.86 5.4 4.19	 44.4 22.9	180 10 115	Sacrificed 11 hr. later
III 16.2	700 c.c. Multiple	150 c.c. 4 × conc. canine plasma		1.0318 1.0325		7.55 8.19	35.2 46.9 32.0	140 85 160 125	Sacrificed 19 hr. later
IV 9.0	364 c.c. Multiple	50 c.c. 4 × conc. canine plasma			14.45 12.0 9.5	6.53 5.34 3.9	41.6 36.4 28.0	150 70 110 85	Sacrificed 3.5 hr. later
V 8.0	377 c.c. Multiple	85 c.c. 4 × conc. canine plasma			12.25 11.4 7.2	5.74 4.31 3.24	37.6 31.1 22.5	135 50 105	Rapid downfall and death after last 30 c.c. Harmful effect
VI 7.5	261 c.c. Multiple	50 c.c. 4 × conc. human plasma; 100 c.c. saline solution by hypo.			12.0 9.3		34.0 21.5	160 40 exp.	Expired suddenly Harmful effect
VII 14.3	505 c.c. Multiple	90 c.c. 4 × conc. human plasma; 120 c.c. saline solution by hypo.		1.0246 1.0215 1.0239	15.85 12.95 9.8		44.0 36.1	155 70 135	Lived
VIII 8.0	305 c.c. Multiple	90 c.c. 4 × conc. human plasma			10.8 10.15 6.5		29.0 28.7 18.8	140 60 115	Sacrificed 3 hr. later
IX 6.5	250 c.c. Two-stage	360 c.c. Normal canine plasma	1.0511 1.0541 1.0476	1.0246 1.0228 1.0249		6.34 6.8 5.0	38.3 42.1 32.2	140 45 105	Lived
X 13.5	615 c.c. Multiple	600 c.c. Normal canine plasma	1.0577 1.0450			8.94 7.5 4.8	39.2 28.4 18.6	190 85 125	Expired approximately 8 hr. later

There were 20 animals in the posthemorrhagic shock group, 16 of which received concentrated plasma and 4, normal plasma. In the traumatic group 6 dogs received concentrated plasma and 3, normal plasma. The volumes of normal and concentrated plasma given were calculated so as to have comparable amounts of protein. In the tables the essential data from 10 animals in the hemorrhagic group and 6 in the traumatic group are given. The data in the other 13 animals were similar to those presented in the tables.

The animals were all in deep shock as demonstrated by the depression in the blood pressure, the collapsed peripheral veins, and the thready and very weak femoral artery pulse. Survival time was not determined and the observations were confined to the interval a few hours after treatment.

Seven of the animals in posthemorrhagic shock showed slight hemocytoconcentration. The others displayed slight hemodilution. In the traumatic shock group hemocytoconcentration failed to occur in only one animal.

It was observed that concentrated plasma caused hemodilution in each case as reflected by a drop in the whole blood specific gravity, a decrease in the red cell count, hemoglobin concentration, and hematocrit. This hemodilution was observed to occur in several instances within five minutes after the injection. During the hemodilution the plasma specific gravity remained about the same, meaning that fluid was withdrawn into the circulation. The elevation in the blood pressure paralleled this hemodilution. It was also observed that the injection of normal plasma with a comparable amount of protein content produced similar results.

As is pointed out in the table, several of the animals responded unfavorably to concentrated plasma. These ill effects were represented by a rigor, lack of elevation of the blood pressure, or even sudden death. These irregular findings could not be understood at first because during the same period very favorable results were being obtained from the use of concentrated plasma in clinical shock.[4, 10]

In subsequent experiments on normal unanesthetized animals[11] definite ill effects were encountered when concentrated plasma was injected rapidly intravenously. There was usually transient but very severe tetany. Most animals subsequently became ataxic, exhibited carpopedal spasm, vomited or retched severely, became lethargic, and occasionally fell asleep. These ill effects would last a varying period of time, and then the animal appeared quite normal. By considering the amount of sodium citrate used as anticoagulant it became apparent that the concentrated plasma contained at least 2 per cent sodium citrate. When such quantities of sodium citrate were given to normal animals similar tetany was observed. As a further check on the citrate effect, it was

TABLE II

TRAUMATIC GROUP

NO. AND WEIGHT (KG.)	METHOD	TREATMENT	BLOOD SPECIFIC GRAVITY	PLASMA SPECIFIC GRAVITY	HB. (GM. %)	R.B.C. (MILLIONS)	HEMATOCRIT (%)	B.P. (MM. HG)	REMARKS
I 8.8	Trauma	100 c.c. 4 × conc. canine plasma	1.0495 1.0646 1.0516	1.0219 1.0367 1.0312		5.46 4.6	32.3 20.0	145 50 120 50	Expired 1.5 hr. after treatment
II 7.0	Trauma	90 c.c. 4 × conc. canine plasma	1.0531 1.0599 1.0455	1.0282 1.0322 1.0281		5.96 6.25 3.46	36.2 17.5	155 80 165	Suddenly expired after B.P. to 165 Harmful effect
III 16.4	Trauma and tourniquet	100 c.c. 4 × conc. canine plasma			14.4 9.95 4.65	3.97 3.55 2.56	22.0 22.0 19.0	130 55 110	Expired 3 hr. later
IV 9.9	Trauma and histamine, 13 c.c. 0.1% solution I.V.	100 c.c. 4 × conc. canine plasma	1.0527 1.0543	1.0280 1.0237		6.64 6.26	29.4 38.2	150 50 120	Expired Harmful effect
V 10.8	Trauma	480 c.c. Normal canine plasma	1.0557 1.0671 1.0442	1.0228 1.0325 1.0232		6.7 7.7 4.5	38.4 32.6 24.0	160 70 110	Sacrificed 2.5 hr. later
VI 9.4	Trauma and histamine, 12.5 c.c. 0.1 % solution I.V.	400 c.c. Normal canine plasma	1.0529 1.0548 1.0456	1.0266 1.0237 1.0224				160 85 120	Sacrificed one hr. later

demonstrated that the administration of neutralizing amounts of calcium chloride with, or shortly before, the concentrated plasma prevented the tetany. Likewise, the administration of concentrated serum without citrate failed to produce tetany. However, the ataxia, retching, vomiting, and lethargic appearance persisted. Since the blood had been collected from donor dogs under nembutal anesthesia, it was assumed that a small quantity of nembutal was being given in the plasma. This assumption was shown to be correct by the production of similar ill effects when small doses of free nembutal were administered intravenously to normal animals. As a final check to these two types of reactions the dog plasma containing citrate-nembutal was dialyzed at low temperature against normal saline solution. After sufficient dialysis the plasma did not produce these ill effects.

TABLE III

Illustrating the effect of citrated dog plasma (at least 2% sodium citrate) containing nembutal when administered intravenously in concentrated form (4 × conc.) to a normal dog under nembutal anesthesia (4 gr. nembutal intravenously).

TIME (P.M.)	AMOUNT INJECTED CONC. CANINE PLASMA	B.P. (MM. HG)	REMARKS
2:47		160	Weight 7.8 kg. Dog under nembutal anesthesia
3:06	10 c.c.	90-140	Wide fluctuations of Hg manometer
3:07	10 c.c.	120-140	Severe tetany began, deep breath, marked rigidity
3:08 to 3:12	55 c.c.	60-165	Tetany persists
3:14		30	Respirations almost ceased, shock-like state
3:32		135	Normal respirations
5:50		140	Appears normal, complete recovery
Total	75 c.c.		

It was then evident that the occasional ill effects in the shock group were due to two causes: First, the rapid administration of a relatively large amount of sodium citrate, which before its oxidation bound sufficient calcium to produce tetany. It is known that such rapid calcium ion deficiency may produce cardiac embarrassment by vagal inhibition or vagal paralysis.[12, 13] Second, the barbiturate effect could be an aggravating factor in animals in deep shock, although not at all dangerous to normal animals. It has been observed that following a favorable blood pressure response in experimental shock therapy the animal may begin to awaken from the comatose state. If at this time a small amount of nembutal is given intravenously there may be a definite drop in the blood pressure. When the animal is in shock these ill effects may be unnoticed and the plasma protein solution may be considered to be ineffective therapeutically. Variations in animal tolerance, variations in the plasma, and the masking effect of the depressed condition of animals in shock may account for the fact that all experiments did not

demonstrate these ill effects. These 50 to 100 c.c. doses in 10 kg. dogs would be comparable to 500 to 1,000 c.c. doses in an 80 kg. person. It is very difficult to administer such huge doses of concentrated plasma to a human being in such a short period of time. Therefore, similar complications in adult human beings would not be expected from the administration of concentrated plasma. However, in children we[4] have observed such calcium binding effect with transient tetany.

DISCUSSION

These studies illustrate the ability of concentrated plasma to produce hemodilution in animals in deep hemorrhagic and traumatic shock. The hemodilution considerably exceeds that which would be expected from the injection of the decreased volume of the concentrated plasma alone. Therefore, there must be an increase in the blood volume resulting from a withdrawal of fluids into the circulation at the capillary beds. This indicates either that the fluid shift occurs through damaged capillaries or that undamaged capillaries are available in deep shock, through which interstitial fluid may be drained into the vascular system.

In shock the therapeutic efficiency of any plasma protein solution depends largely on its protein content, since it is the protein molecule that maintains the plasma volume. Hence, an equal degree of hemodilution is obtained when normal or concentrated plasma of comparable protein content is given to different animals at similar rates of injection. These observations were first mentioned by Magladery, Solandt, and Best.[14] However, the action of these two agents is different, for, whereas concentrated plasma or serum causes an internal shift of fluids, a normal plasma injection merely adds more fluids. Recent evidence suggests that cells take up water in experimental hemorrhagic[15] and freezing shock.[5] The possibility that concentrated plasma may reclaim such water[11] appears very logical. It seems that the withdrawal of misplaced fluids by the use of concentrated plasma is more of a reversal of certain shock mechanisms than would be expected from the external addition of normal plasma.

It may be stated then that the efficacy of any protein solution in the treatment of shock depends upon the total protein content; the protein concentration, which determines its ability or lack of ability to shift fluids from an extravascular to an intravascular position; and its rate of administration.

It has recently been stated[16] that large volumes of dilute plasma are more beneficial in the treatment of shock than normal or concentrated plasma, and a failure of response was observed in certain animals given concentrated plasma. This question is discussed in other publications.[5, 17] Suffice it to mention at present that plasma protein solutions varying in protein concentration should be compared not only ac-

cording to comparable volumes and comparable protein content, but also according to rate of injection. Whether the plasma contains sodium citrate or barbiturate should be mentioned. Obviously, if a deleterious substance is present in the plasma, its injection may be innocuous in dilute form due to the slowness of administration, and quite harmful in concentrated form because a greater quantity of the substance is ordinarily given per unit of time.

The term "four times concentrated plasma" signifies that the dry product was dissolved in one-fourth of the original volume. The protein content of such a concentrate depends on the protein concentration of the original citrated plasma prior to desiccation. In spite of the resulting variation in the protein content of the original citrated plasma, the final concentrated product is usually loosely compared with the osmotic property of normal plasma taken to contain 7 Gm. per cent of proteins. Such terminology has given rise to much confusion. It seems essential to drop the term "four times concentrated plasma" and speak directly of its protein content in grams per cent. Unfortunately, in the present studies we failed to take these considerations into account. As a result of this, certain disparities appear in the data of this report. For instance, when the original blood volume is calculated on the basis of body weight and subsequent volume changes are calculated according to changes in the hemoglobin concentration or hematocrit, it is revealed that the volume increases following the concentrated plasma injections are smaller than would be expected from the injection of true four times concentrated plasma (approximately 25 Gm. per cent). Obviously the concentrated plasma solutions used in the present studies contained smaller amounts of protein than might be expected from the use of the term four times concentrated plasma, which is used to define a solution obtained by dissolving the solid residue in one-fourth of the original volume. Assuming that all of the plasma given remained within the blood stream after the equilibration of its protein content, an analysis of the data from two experiments might clarify these points. Following is an example:

Dog No. VIII

Weight	8	kg.
Calculated blood volume (9% body weight)	720	c.c.
Control hematocrit	29.0	%
Control red cell volume	208.8	c.c.
Volume red cells bled	88.5	c.c.
Shock red cell volume	120.3	c.c.
Shock blood volume (120.3 ÷ 28.7)	419	c.c.
After treatment blood volume (120.3 ÷ 18.8)	640	c.c.
Increase in plasma volume	221	c.c.
Expected increase 90 × 4	360	c.c.

On the same basis dog No. III showed a calculated volume increase of 267 c.c. out of an expected increase of 600 c.c. Such calculations are influenced by other factors, and therefore are not strictly accurate but

can be used to demonstrate the relative trend in the experiments and show that the volume increase was much greater than would be expected from the volume of concentrated plasma alone. This method of calculating volume changes cannot be used in the traumatic shock group because the volume of red cells lost is a totally unknown quantity.

In a subsequent report[5] it is shown that in the treatment of experimental shock with concentrated serum the plasma volume increase is directly proportional and equivalent to the grams of protein injected.

Most workers in this field are willing to agree that concentrated plasma given in standard glass syringes can be handled more rapidly and with greater ease than other more bulky plasma solutions, such as normal or dilute plasma. However, its therapeutic efficiency is questioned, the dangers of reactions are emphasized, and it is stated that dehydration may be a harmful sequel. Therapeutically concentrated plasma is capable of producing hemodilution in direct proportion to its protein content. A plasma that is free from agglutinins, bacteria, and pyrogens and that has been prepared by desiccation from the frozen state, in our experience[10] will not produce reactions when given in concentrated form. Dehydration following the injection of concentrated solutions depends on the type of solution given. For instance, when concentrated glucose is given intravenously it draws fluids from the tissues and thus increases the plasma and blood volume. At the same time because the plasma is diluted its composition is altered. The kidneys, being more concerned with plasma composition[18] rather than volume, reject the added water in the urine. The glucose is either taken up by the liver or metabolized. Therefore, water is lost from the body, the glucose is removed from the blood stream, and dehydration may eventually result.[19] When concentrated plasma is injected it draws fluids from the tissues but the plasma composition remains about normal. The kidney will not reject the added fluid readily and the mechanisms of dehydration are thus avoided. In nephrosis or low protein edema the diuresis seems to be related to a decrease in sodium intake plus a greater renal plasma flow that is associated with plasma therapy.

Concentrated plasma should receive more consideration in the treatment of shock. It has a definite physiologic basis for its rationale. The ease of handling gives it tremendous military advantages.

CONCLUSIONS

1. Concentrated plasma produces hemodilution rapidly in severe posthemorrhagic and traumatic shock in dogs.

2. The hemodilution is due to the shift of fluids from an extravascular to an intravascular location as shown by the lack of change in plasma specific gravity.

3. Normal plasma with comparable protein content is capable of producing hemodilution.

4. The difference in the action of concentrated and normal plasma is pointed out.

5. Two distinct causes for untoward reactions in experimental animals after the injection of concentrated plasma are discussed.

6. The importance of concentrated plasma in military practice is reemphasized and certain objections by other workers are briefly discussed.

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A MODIFIED TECHNIQUE OF SURGICAL LIGATION OF PATENT DUCTUS ARTERIOSUS

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IN 1939, Gross and Hubbard[2] reported the first successful ligation of a patent ductus arteriosus. Stimulated by this brilliant surgical achievement, the operation soon was attempted by others with the result that the effectiveness of ductal ligation in correcting circulatory abnormality quickly became established. The technique described by Gross[1] has been generally employed up to the present time, and apparently has proved quite satisfactory. The chief dangers which attend operation are (1) hemorrhage as the result of inadvertent injury of the ductus or pulmonary artery, and (2) injury of the left recurrent laryngeal nerve. Hemorrhage is most apt to occur as the posteromesial wall of the ductus is being separated from the adjacent, main trunk and right branch of the pulmonary artery. (This portion of the ductal wall faces away from the operator, and therefore cannot be visualized completely.) Thus, its separation from the pulmonary artery constitutes a relatively "blind" procedure, during which either of the afore-mentioned structures may be inadvertently torn. In an attempt to avoid such accidents, ductal dissection of necessity must be performed carefully and unhurriedly. (Gross has reported a case in which almost two hours were spent in performing this step of the procedure, alone.) Gross has stressed the necessity for careful and complete exposure of the left recurrent laryngeal nerve, as a guide to the location of the ductus. If injury to the nerve is to be avoided, considerable time may have to be expended in this dissection also. The operation for ligation of patent ductus arteriosus therefore is one which, in general, is apt to be time consuming if serious complications are to be avoided.

In 1940, Vesell and I[3] reported the first case of subacute bacterial endarteritis complicating patent ductus arteriosus, in which recovery followed surgical treatment. During the same year we reported our experiences in the surgical treatment of three additional cases,[4] and emphasized that in the presence of infection the likelihood of accidental hemorrhage is increased considerably. The latter is attributable to two important pathologic features which may accompany the superimposed infection: (a) friability of the ductus, as the result of atheromatous and infectional disease changes; and (b) periarteritis, which renders the ductus very firmly adherent to the surrounding structures, the most important of which is the pulmonary artery. Furthermore, since our four

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patients were adults, and the duration of the congenital lesion therefore longer, the pulmonary artery was relatively more dilated, thin walled, and susceptible to accidental injury than in the case of children. Thus in spite of the fact that great care was exercised in dissecting out and manipulating the ductus, hemorrhage occurred in three instances. Of these, two patients succumbed upon the operating table. In the third, profuse bleeding from a tear in the ductus was successfully controlled. (The last patient recovered and has been entirely free of all evidence of subacute bacterial endarteritis since operation, almost two years ago.)

With the realization that we were dealing with a disease which still is considered to be almost invariably fatal under conservative forms of therapy, I was encouraged to continue my surgical efforts, despite the fact that operation had proved to be so hazardous. It was my hope that the latter might be rendered more safe by modifying the operative technique; and accordingly, when the next patient with patent ductus arteriosus complicated by subacute bacterial endarteritis was presented for surgical treatment, a somewhat different type of procedure was carried out. Shortly thereafter another infected case was operated upon, with the same technique. Both of these patients apparently have been cured of the bacterial infection;[5, 6] in neither case was the surgical procedure attended by hemorrhage and the time consumed in operating was reduced materially. (In the latter case, operation was performed in fifty-five minutes). Since the avoidance of hemorrhage and the shortening of operation appear to be attributable chiefly to the modified technique, the latter seems worthy of description. It is applicable not only to infected cases but also to uninfected cases, and is based upon a number of principles which themselves are dictated by certain anatomic and pathologic findings. The latter are as follows: (a) the aorta is by far the strongest structure and the one most resistant to injury in the immediate operative field; (b) the aortic end of the ductus is normally the strongest portion of the ductus and the portion which is least apt to be affected by atheromatous and infectional changes; and (c) it is easier and safer to free a vessel which is firmly adherent to some surrounding structure by carrying out the procedure in a plane of cleavage within the adventitia, than by attempting to strip the intact vessel from the surrounding tissues.

In sequential order, the steps of the modified operation consist of: (a) Entering an inter- or subadventitial plane of dissection, upon the aorta, close to the aortic end of the ductus; (b) "tunnelling," within the above plane (upon the aorta), completely behind the aortic end of the ductus; (c) developing the plane from the aorta, to the posteromesial wall of the ductus itself.

The procedure is performed under rather complete visualization, and by keeping within the adventitial plane of the aorta and adjoining ductus, injury to the pulmonary artery and recurrent laryngeal nerve is minimized.

The operative entry of the thorax and the exposure of the aortic arch and pulmonary artery are performed in the manner described by Gross. (Although Gross has recommended entry of the thorax routinely through the second intercostal space, in certain cases the lower border of the aortic arch is seen in the x-ray film to lie at or near the level of the third interspace. Under such circumstances, an incision in the third interspace affords a more direct approach to the ductus.) The upper end of the trunk of the dilated pulmonary artery is identified and dissected from the aortic arch. After this has been accomplished, the position of the ductus is determined by palpating for the point of maximal thrill in the region of the concavity of the aortic arch. The pulmonary artery, which in adults often completely obscures the aortic arch from view (Fig. 2),

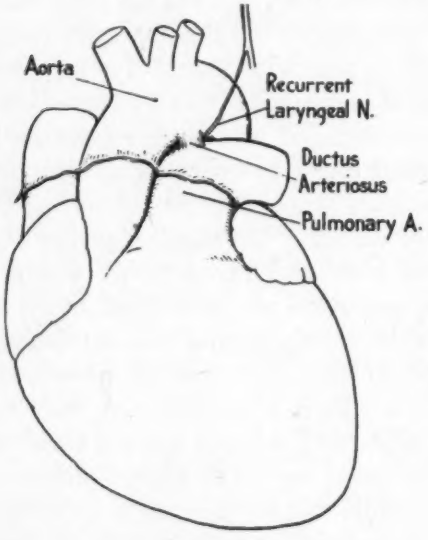

Fig. 1.

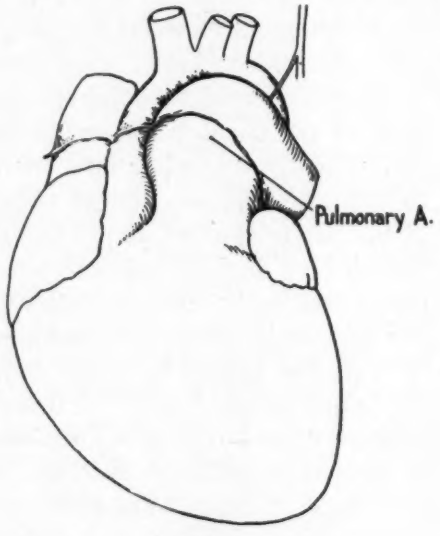

Fig. 2.

Fig. 1.—Schematic drawing to illustrate relative size of the aorta, pulmonary artery, and ductus arteriosus, shortly before birth.

Fig. 2.—Schematic drawing to illustrate the findings in cases of patent ductus arteriosus of long duration (as in adults). Note the marked dilatation of the pulmonary artery. (The latter often obscures the aorta completely.) The ductus arteriosus is not visible.

then is gently displaced downward as far as possible and held in this position by a ribbon retractor (Fig. 3). This exposes the general region of the ductus. The anterolateral surface of the ductus next is exposed by removing the overlying areolar tissue and lymph nodes. The angle between the upper surface of the ductus and the aorta, and the angle between the lower surface of the ductus and the aorta then are exposed carefully in the same manner. Care is taken to avoid the proximal portion of the recurrent laryngeal nerve as it leaves the trunk of the vagus near the lower of the two angles mentioned above. The adventitia of the aorta at the upper angle (Fig. 4, *A*) is grasped with a toothed forceps close to the ductus, and incised with fine scissors. A fine, curved, cystic duct clamp then is passed into the incision in the aortic adventitia

and advanced slowly and progressively, downward and laterally, along the posterior wall of the aorta immediately adjacent to the aortic end of the ductus. As this plane is developed, a narrow ribbon retractor is in-

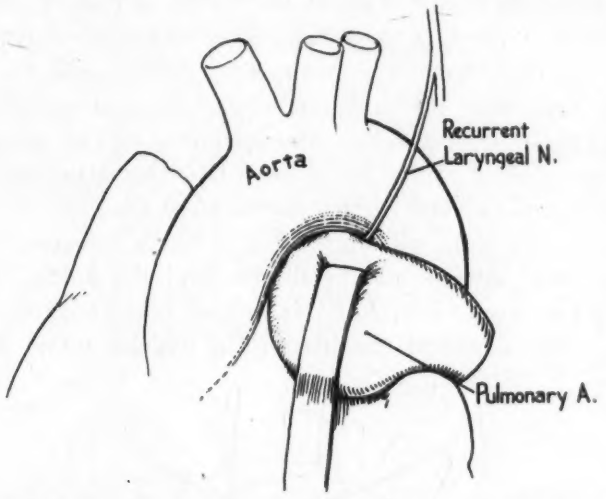

Fig. 3.—First stage of the author's modified operative procedure. The pulmonary artery has been dissected from the aorta and is displaced downward by a retractor. After removing areolar tissue and lymph nodes from the region of the concavity of the aortic arch, the ductus arteriosus comes into view.

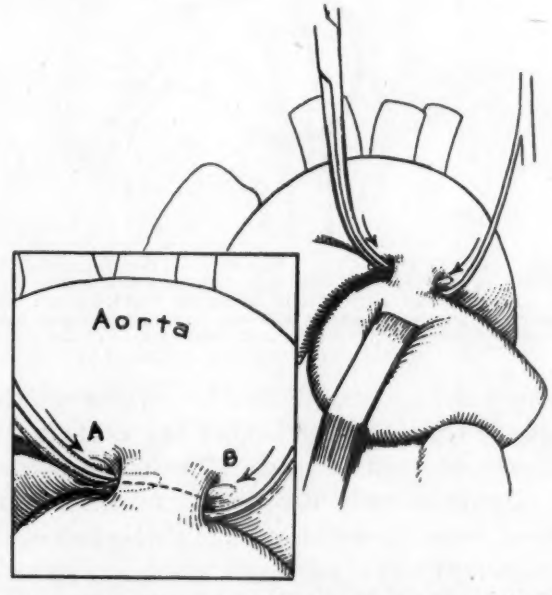

Fig. 4.—Operation continued. An incision is made in the aortic adventitia at *A*. A fine curved cystic duct clamp is introduced into the adventitial incision and gradually passed downward and laterally (upon the aorta) as far as possible. A similar dissection is begun at *B* and carried upward until a "tunnel" has been completed (beneath the adventitia of the aorta) behind the aortic end of the ductus.

troduced; and the aortic adventitial tissue, which has been stripped free, is retracted medially. The origin of the right branch of the pulmonary artery, which lies outside (mesial to) the freed adventitia thus

is retracted automatically (Fig. 5). Once the plane of cleavage has been entered properly, tiny gauze sponges held in small curved hemostats are employed as atraumatic dissectors. As the dissection progresses along the posterior wall of the aorta, it also is extended for a short distance behind the ductus. The retractor which holds the stripped adventitial tissue and pulmonary artery aside is advanced progressively deeper as the posteromesial surface of the aortic end of the ductus is freed. As a result, the stripping of this portion of the ductus is accomplished under fairly complete visualization. After the separation of the aortic end of the ductus has been carried from above downward as far as room will permit, attention is directed to the angle between the lower surface of the ductus and the aorta. The aortic adventitia in this area (Fig. 4, *B*) is incised near the ductus. As the assistant holds the edges of the adventitial incision apart, the curved,

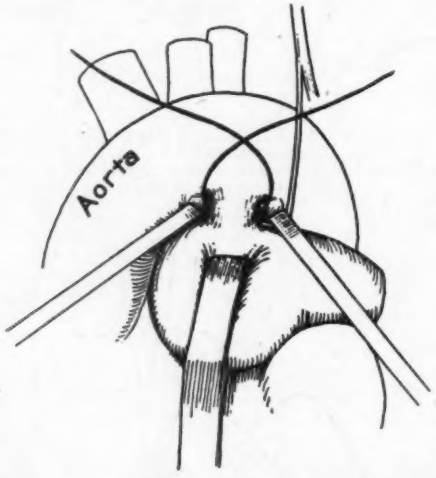

Fig. 5.—After the "tunnel" within the adventitia of the aorta has been completed, the plane of dissection is carried onto the posteromesial surface of the ductus. Note that the retractors within the adventitia hold aside the right branch of the pulmonary artery and the recurrent laryngeal nerve without actually coming into contact with either of those structures. The ligature has been passed behind the ductus.

cystic duct clamp previously mentioned is introduced into the proper plane and passed gently upward behind the aorta adjacent to the ductus. The dissection then is extended onto the aortic end of the ductus, in the manner already described. As the adventitia, which has been separated from the aorta and the aortic end of the ductus, is retracted, the recurrent laryngeal nerve which lies immediately below and outside it is automatically held aside without coming into actual contact with the dissecting instrument (Fig. 5). A small ribbon retractor once more is introduced into the upper adventitial incision as deeply as possible from above downward, and by retracting the freed adventitia and subjacent pulmonary artery mesially, the posteromesial surface of the ductus again is exposed. The tip of the curved clamp which is being directed from below upward, behind the

ductus, now can be readily visualized and the freeing of the posteromesial ductal wall completed. In ligating the ductus (Fig. 6), two heavy silk ligatures are employed. One of these is placed directly adjacent to the aorta and the second nearby. In view of the minimal infectional changes and the greater thickness of the ductal walls in this area, accidental tearing of the ductus during ligation, and cutting through of the ductal ligatures, subsequently, is minimized. Furthermore, a ligation of the ductus very close to the aorta has the additional advantage of excluding infected vegetations within the interior from direct contact with the aortic blood current. (The role of surgical ligation in the cure of infected patent ductus will not be set forth here, but will be discussed in detail in a forthcoming paper which will deal with the rationale of surgical treatment.)

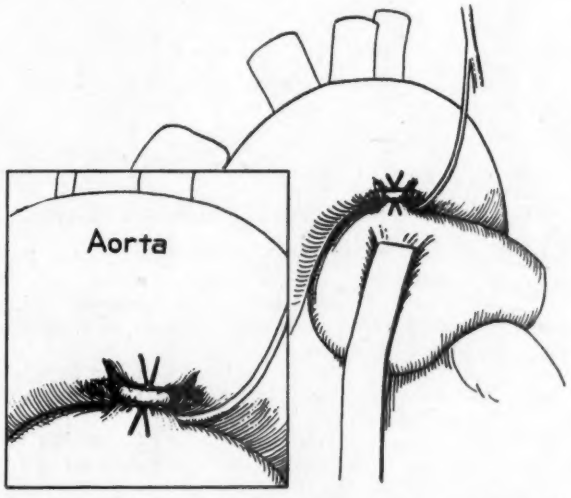

Fig. 6.—Final stage of procedure. Double ligatures tied about the ductus, as close to aortic end as possible.

Following ligation of the ductus, the local site is sprinkled with 1 to 2 Gm. of powdered sulfathiazole or sulfapyridine. The mediastinal pleura is left open, in order to permit fluid exudate to drain freely into the pleural cavity. The thoracic cage and parietes are closed in layers, the lung being inflated by the anesthetist just as the closure of the pleural cavity is completed.

SUMMARY AND CONCLUSIONS

1. The chief dangers attending operation upon patent ductus arteriosus are (a) hemorrhage as the result of inadvertent injury of the ductus or pulmonary artery, and (b) injury of the left recurrent laryngeal nerve.

2. In the presence of subacute bacterial endarteritis, the likelihood of accidental hemorrhage may be greatly increased as the result of friability

of the ductus and its firm adherence to the surrounding structures, especially the trunk and right branch of the pulmonary artery.

3. In order to minimize the likelihood of injuring the ductus, pulmonary artery and recurrent laryngeal nerve, the original operative technique described by Gross, has been modified.

4. These modifications consist of: (a) entering an inter- or subadventitial plane of dissection, upon the aorta, close to the aortic end of the ductus; (b) "tunnelling," within the above plane (upon the aorta), completely behind the aortic end of the ductus; (c) developing the plane from the aorta to the posteromesial wall of the ductus itself.

5. Of four operative cases of patent ductus arteriosus complicated by subacute bacterial endarteritis reported originally, serious hemorrhage occurred in three. In two recent similar cases operated upon with the technique herein described, hemorrhage did not occur and the time required for operation was reduced materially.*

6. The improved technique is applicable both to infected and uninfected cases of patent ductus arteriosus and should reduce the hazards of operation considerably.

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*Since submitting this paper for publication, two more cases of infected patent ductus arteriosus have been operated upon successfully.

LIGATION OF A PATENT DUCTUS ARTERIOSUS: REPORT OF A SUCCESSFUL CASE

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THE ductus arteriosus in fetal life serves the important function of shunting blood from the pulmonic artery to the aorta, thus by-passing the lungs. Incomplete expansion of the lungs produces a high resistance in the pulmonary vascular bed. When the child is born and the lungs expand, the peripheral pulmonary resistance to blood flow is lowered. Pressure in the aorta then is higher than pressure in the pulmonary artery. The ductus normally closes and all the blood in the pulmonary artery passes through the lungs to be aerated. If the ductus arteriosus fails to close, a reversal of flow takes place, and blood passes from the aorta to the pulmonary artery through the ductus. The patient then possesses what is essentially an arteriovenous aneurysm.

Patten[1] claims the degenerative intimal changes take place in the ductus arteriosus during the latter part of fetal life, and it is his belief that increasing amounts of blood flow through the lungs even before birth.

Christie[2] studied a large series of routine autopsy specimens from infants to determine when the ductus arteriosus is normally obliterated. In infants of one month, an open ductus was found in 44 per cent; at two months it was open in 12 per cent; and at eight months it was open in 2 per cent. In a small percentage of cases, the ductus remained open permanently.

Barclay[3, 4] and his co-workers performed detailed x-ray studies on lambs delivered by Cesarean section, as well as on adult animals. They concluded that the ductus closes functionally within five minutes after delivery. The x-ray technique of visualizing the ductus arteriosus does not seem applicable to clinical use at present.

Gross[5] sets an arbitrary time limit of one year, believing that an open ductus after that time should be regarded as abnormal. He also considers the etiology of a patent ductus arteriosus and discusses three interesting possibilities. The position and direction of the vessel is important. Usually it enters the aorta at an acute angle. As the acuteness of the angle decreases, the ductus is more and more exposed to intra-aortic pressure. If the angle becomes a right or even an obtuse angle, the force of intra-aortic pressure can readily be exerted to maintain patency of the lumen of the ductus. In his operative experience, Gross

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has repeatedly observed this deviation from the usual acute angle. The second possibility is a defect in the elastic fiber of the media, decreasing the contractibility of the ductus. A third possibility is a deficient neurovascular tonus. Gross points out that the discussion of these possibilities does not apply to cases in which another congenital defect acts to maintain an open ductus arteriosus as a compensatory mechanism.

PATHOLOGIC PHYSIOLOGY

Hazards of a Patent Ductus Arteriosus.—The patent ductus arteriosus may be accompanied by other congenital abnormalities. Such pathologic combinations come within the scope of this presentation only as considerations in the differential diagnosis of patent ductus arteriosus. A patent ductus arteriosus can and does exist as a single congenital cardiac anomaly. The hazards of its presence alone are sufficient to justify operation in selected cases.

Eppinger and Burwell,[6] with Gross,[7] studied the effects of the patent ductus on the heart and circulation in six patients before and after ligation of the ductus. They pointed out that in the presence of a patent ductus arteriosus, the flow is from the aorta to the pulmonary artery. No flow from the pulmonary artery to the aorta takes place, and hence the patient is not cyanotic. From blood samples taken from the vessels both before and after ligation, they estimated that the volume of blood flowing from the aorta to the pulmonary artery varied from four to nineteen liters per minute. In other words, the shunt diverted from 45 to 75 per cent of all blood pumped into the aorta by the left ventricle. They also found that the left ventricle expelled from two to four times the volume of blood expelled by the right ventricle over a given period of time. Obviously the adjustment of the circulation to the patent ductus arteriosus is made by an increase in left ventricular output. If compensation is incomplete, there may be a diminution of blood flow to the periphery. These studies clearly show the tremendous strain a patent ductus imposes upon the heart.

The hazards of a patent ductus arteriosus are well demonstrated by studies of the life expectancy of such patients. Maude Abbott[8] cites ninety-two patients in whom the mean age of death was twenty-four years. Since the series includes twenty patients who died in infancy, the mean age of death is somewhat confusing.

Jones, Dolley, and Bullock[9] reviewed seventy-six cases from the literature and added four from personal experience, a total of eighty cases. All patients were over three years of age at the time of death, and the diagnosis was proved by autopsy in each instance.

By fourteen years, 14 per cent died of their heart lesion.
By thirty years, 50 per cent died of their heart lesion.
By forty-one years, 71 per cent died of their heart lesion.

In analyzing the causes of death in these cases,

18, or 23 per cent, died of congestive heart failure.

5, or 6 per cent, died of rupture of the ductus or left ventricular failure.

42, or 53 per cent, died of bacterial endocarditis.

4, or 5 per cent, died of an associated cause of death in which the cardiac state was contributory.

Maude Abbott[8, 10] found the incidence of subacute endocarditis to be 25 per cent. Gross[11] regards it as 20 to 25 per cent.

The hazards of the patent ductus arteriosus are cardiac decompensation (prognosis here when untreated is probably directly related to the size of the opening), bacterial endocarditis or endarteritis, aneurysmal dilatation of the ductus,[12] rupture of the ductus,[9] and possibly retrograde thrombosis with release of emboli to various parts of the body.

DIAGNOSIS

During the first three to four years of life, the patent ductus arteriosus is seldom recognized. In some cases, during an incidental physical examination, the murmur may be heard, and the child may be given the "waste-basket diagnosis" of congenital heart disease. When the child progresses to the period of increasing exercise, symptoms of dyspnea, increased heart rate, or a pounding heart may develop. Some patients complain of a buzz or a hum in the chest. Decompensation is unusual in children. Cyanosis is present only as a terminal symptom of impending death. As a result of pressure on the recurrent nerve, hoarseness may be present. According to Gross, the lesion is more frequently found in females than in males.

The physical findings boil down to a few important signs and numerous subsidiary signs of confirmatory evidence. Probably the most important single sign is a continuous coarse machinery murmur found at the pulmonic area in the second or third interspace to the left of the sternum. The murmur is accentuated in systole. It is transmitted to the pericardium and to the axillae, especially the left axilla. It is often heard over the back, and is diminished over the neck vessels. A palpable thrill often accompanies the murmurs. According to Bullock, Jones, and Dolley,[13] when infants and patients with cyanosis are excluded, such a murmur is the strongest evidence for the presence of a patent ductus arteriosus without other congenital defects which would contraindicate operation. Patients with multiple defects rarely survive infancy. Cases with aortic or pulmonary atresia, where the ductus acts as a compensatory mechanism, show cyanosis. Defects, such as a patent interventricular septum, a patent foramen ovale, or a bicuspid aortic valve may exist under the conditions mentioned, but they would not change the indication for operation. Coarctation of the aorta should be carefully ruled out. A second sign of importance is the blood pres-

sure. The systolic level is normal or slightly lowered, while the diastolic level is greatly decreased. With the high pulse pressure, the Corrigan pulse and the pistol-shot sounds may be detected in the peripheral arteries. An appreciable number of patients are below par in weight and physical development, although they are mentally alert.

Inspection may show visible pulsation with increased thrust. Percussion shows a heart of normal or slightly increased size. The laboratory data are not remarkable. The electrocardiogram is normal, a point of differentiation from pulmonary stenosis which shows right axis deviation.

The x-ray findings are not diagnostic, but they serve as important contributory evidence. They also bear witness as to the effectiveness of surgical therapy.[6] In roentgenograms, kymograms, and fluoroscopy, one should look for the following:

1. A possible enlarged left ventricle.
2. Increased pulsation of the left ventricle and the pulmonary artery.
3. Unusual prominence of the pulmonary artery.
4. Engorgement of the pulmonic vessels.
5. Increased pulsations of pulmonary artery branches.
6. Possible dilation of the left auricle.

SELECTION OF CASES

In one of his early papers, Gross[5] stated the factors which guided him in the selection of cases for operation. In the first place, there should be no associated congenital cardiac defects. A septal opening or a mild (rheumatic) mitral stenosis are lesions which do not contraindicate operation. In the second place, he believes the patient should show evidence of impaired cardiac function which is unlikely to correct itself by spontaneous closure. To meet the second requirement, the patient must be observed over a period of time. In addition, there should be the assurance that a patent ductus exists as evidenced by the presence of a characteristic murmur, thrill, blood pressure, and x-ray signs as stated.

Gross also stated that the operation should not be done in the presence of bacterial endocarditis. Touroff and Vessell,[14] feeling that ligation of the ductus might benefit the patient with a patent ductus who also had endocarditis, operated upon four cases. Operation in the first case was successful and the patient remained cured for thirty-six weeks, at which time the paper was published. The second patient succumbed to the disease, and the third and fourth patients died of hemorrhage on the table.

OPERATION

R. E. Gross, of Boston, deserves and unquestionably will receive lasting recognition for his excellent work in devising an efficient operative method for ligation of the ductus arteriosus. The careful work of Gross

and his colleagues, the thoroughness of investigations, the manner with which all presentations are made, and the strict selection of cases distinguish all their presentations.[5, 11, 15, 16]

Munro,[17] in 1907, first suggested the possibility of ligating the ductus arteriosus, advocating a transsternal approach. According to Gross, O'Shaughnessy[18] was the first to make the attempt, but it was not successful. Strieder[19] made the next attempt in a patient who had subacute bacterial endocarditis, but obliteration of the vessel could not be achieved. It remained for Gross to be the first to perform the operation successfully.

Some of the operative complications are as follows:

1. Hemorrhage from the ductus during dissection.
2. Sepsis.
3. Pneumonia.
4. Inability to identify the duct.
5. Incomplete occlusion of the duct.
6. Perforation of the duct by erosion from the ligature.
7. Injury to the recurrent laryngeal nerve. (This, however, would not be an important matter because the larynx would readily accommodate itself to the injury of this nerve.)
8. Postoperative thrombus.

Postoperatively, Gross observed a cessation of the thrill, disappearance of the murmur (except in a few early cases where the single ligature apparently was not placed tightly enough, and residual murmurs were found), a definite rise of diastolic blood pressure, a diminished cardiac activity, a gain in weight and strength in the retarded children, a return of the cardiothoracic ratio to normal, and cessation of bouts of cardiac failure in two patients who exhibited them preoperatively.

The operative technique of other authors differs from the plan proposed by Gross only in details. Jones and Dolley[21] also operated on thirteen patients with one fatality. In order to allow serous drainage, the mediastinal pleura is not closed. Pleural effusions are aspirated. They follow the teaching of Mont Reid[22] in the selection of ligature material for ligating this large vessel, and recommend an umbilical tape ligature of woven silk $\frac{5}{32}$ of an inch in width. More recently, Gross has been supplementing the ligation of the vessel by means of woven silk with a ligature of cellophane, weight 300, of the unsurfaced type. Herman Pearse, of Rochester, N. Y., has shown by experimental work that cellophane markedly increases the proliferation of fibrous tissue of a vessel so treated. These authors point to Holman's work[23] on arteriovenous fistula where he showed that the obliteration of such a fistula causes an automatic rise of blood volume in the arterial tree. Hence, they feel that postoperative infusions and transfusions should not be used. Their one fatality was a case of postligation thrombus.

CASE REPORT

The patient D. F., a girl aged 15 years, was first admitted to the Santa Rosa Hospital, San Antonio, Texas, May 6, 1941, on the medical service of Dr. Merton M. Minter. Her complaints on admission were cough, headache, weakness, and dyspnea at all times.

Present Illness.—The child was known to have had a heart murmur since the third day of her life. At times she became cyanotic, particularly on marked exertion and after coughing. As a rule she had been able to carry on her usual activities and play, but in the last few years she had gradually noticed more discomfort and respiratory distress. A pounding palpitation had been present as long as she could remember. Her general development had been retarded, more so within the last four or five years. Otherwise, she had been quite well.

Past History.—Previous to the first hospitalization, the patient had measles, chicken pox, and diphtheria. She had no operations and other than the conditions mentioned, she has enjoyed reasonably good health. Menses have not begun.

Physical Examination.—The patient is a pallid, thin, white female of about the stated age of fifteen years. She was somewhat dyspneic and slightly cyanotic. There were no outstanding abnormalities of the head, neck, eyes, ears or throat.

On examination of the chest there was limitation of expansion on the right. The right lung was dull to percussion throughout, with the possibility of fluid at the right base. There was a loud, rough, machinelike murmur in the left second and third interspaces with a palpable thrill. The murmur was audible over the entire precordium, but its greatest intensity was in the left second interspace. The temperature on admission was 101° F.

Fig. 1.—Preoperative electrocardiographic tracing. These changes are not characteristic of any definite form of heart disease. Myocardial damage is indicated.

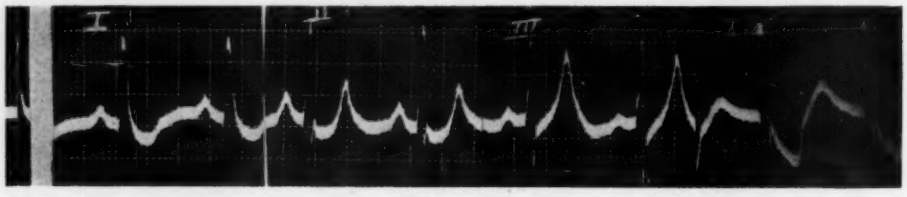


Fig. 2.—Postoperative electrocardiographic tracing. A very mild digitalis reaction is seen. No other electrocardiographic changes are noticed which were not present prior to the operation. Note slowing of the heart rate.

Laboratory Findings.—The urine showed an acid reaction; the specific gravity was 1.020; there was a trace of albumin; the pus was 1 plus; and there was an occasional red blood cell in the microscopic study. The red blood cell count was 4,350,000; the white blood cell count was 10,350; the differential showed 75 per cent polymorphonuclears, 21 per cent small lymphocytes, 1 eosinophile, 3 transitional cells, and 10 stab forms. The hemoglobin was 84 per cent; the coagulation time was 4.5 minutes.

X-ray of the chest showed congestive changes in each lung with increased density above the hilum in the right lung. The heart shadow was displaced well to the left

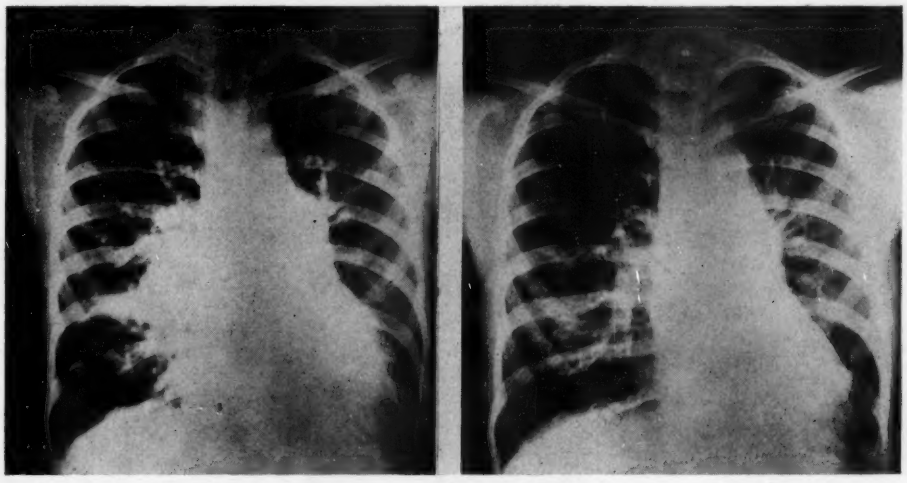
so that the apex was one-half inch from the left lateral chest wall. The pulmonary conus showed marked enlargement.

An electrocardiogram taken at this time showed changes not characteristic of any specific heart lesion, but was indicative of cardiac damage.

The patient obviously had an upper respiratory infection plus congestive failure. A diagnosis of patent ductus arteriosus was first made by Dr. Cresswell, and later confirmed by Dr. Merton M. Minter and Dr. Henry N. Leopold. Treatment with sulfathiazole was instituted for the respiratory infection. Digitalis and salyrgan were given for the congestive failure. She improved rapidly under the regime and on May 20, 1941, was able to go home for further rest.

Second Admission.—Patient re-entered the hospital on June 26, 1941, for the purpose of further study and for surgery. The laboratory findings were essentially the same as before except that the white count had fallen to 8,600. The physical examination was the same as before except that the cyanosis and dyspnea were less marked, and the patient was fever free. The blood pressures taken in each arm and each leg were as follows: Right arm, 118/50; left arm, 114/48; right leg, 160/20; left leg, 165/50.

Operation.—The patient was operated on June 24, 1941. She was placed on her back with a small pillow under the left shoulder, and with the left arm placed above her head. Cyclopropane anesthesia was administered by the intratracheal method. An incision was made on the left side of the chest, beginning at the



A. *B.*

Fig. 3.—Pre- and postoperative roentgenograms. *A.* Note enlarged ventricle, prominent pulmonary conus, and congestion of hilus of right lung. *B.* Note disappearance of the above signs.

lateral border of the sternum and extending laterally to the anterior axillary line over the right second costal interspace. The skin was incised, and the pectoralis muscles were cut in the line of the skin incision. The cartilages of the second and third ribs were cut just medial to the costocartilage junction. The pleural cavity was opened through the second interspace. The lung was collapsed and retracted posteriorly and downward, exposing the pericardium and mediastinum, along which could be seen coursing the phrenic nerve. The mediastinal pleura was picked up gently with fine forceps and was opened through an incision about two inches in length, beginning at the base of the lung and extending upward toward the neck. The fatty areolar tissue was carefully dissected away, exposing the pulmonary artery and the aortic arch. Further dissection exposed the vagus nerve and the

recurrent laryngeal nerve which was picked up and traced medially until it disappeared under the ductus. The thrill at its maximum was found to be over a vessel which extended from the pulmonary artery to the aortic arch, entering the aorta just opposite the left subclavian artery. This structure was four millimeters in diameter and about three millimeters in length. It was carefully freed from the surrounding tissues by blunt dissection until the anterior, medial, and lateral surfaces were freed and thoroughly exposed. When this structure was pressed between the thumb and index finger, the thrill of the heart immediately disappeared. A large aneurysm needle was carefully passed under this structure, separating it from

Fig. 4.—Photograph of patient D. F. illustrating her frailness. Note incision in front, left anterior chest.

the right bronchus. The lumen of this vessel was closed by traction on the silk which had been placed around it, and the condition of the patient was observed for a period of four or five minutes. During the time that the vessel was held closed, the blood pressure in the arm rose fifteen points and the patient exhibited no untoward symptoms. When the traction was released, the thrill reappeared in the pulmonary artery and the auricle of the heart, and the blood pressure again dropped. It was felt this was sufficient evident to prove that the structure with which we were dealing was the ductus arteriosus, and it was accordingly doubly ligated with No. 8 braided silk. A small amount of oozing was encountered in the

region of the recurrent laryngeal nerve, but this was controlled by pressure with moist gauze. No effort was made to close the mediastinal pleura because it was felt that if any oozing should occur it would be better to have it enter the thorax rather than to be held in the mediastinum. The lung was then inflated by positive pressure. The cartilage was sutured with No. 32 annealed wire. Two lengths of size No. 30 wire were passed around the second and third ribs, pulling them into apposition with each other. With continuous No. 1 chromic catgut lock sutures, the soft tissues were closed. The skin was closed with a continuous silk lock suture.

Following the operation, the patient was taken to the x-ray room and the chest fluoroscoped. The upper lobe of the lung was well collapsed; the lower lobe, partially collapsed. There was an estimated 300 or 400 cubic centimeters of air just above the diaphragm. The thrill of the heart had entirely disappeared.

Postoperative Course.—On the first postoperative day, the pulse was 128, but no murmurs were discernible and the thrill was entirely absent. The patient's respirations were 24. She complained a great deal of pain when awake but was somnolent most of the time. On auscultation, breath sounds were markedly diminished at the left base. Percussion was hyperresonant on the left, indicating the presence of left pneumothorax. An x-ray picture taken at this time revealed the right lung to be relatively clear. There was shown an increased density over most of the left lung extending from the second rib anteriorly to the base. Considerable emphysema was present in the soft tissues surrounding the chest wall. The film made with the patient lying on the right side showed the presence of pneumothorax. The blood pressures taken in each arm and each leg were as follows: Right arm, 125/85; left arm, 125/85; right leg, 135/90; left leg, 120/80.

On the second postoperative day, the patient was very restless and cyanotic most of the day. A thoracentesis was done and 720 cubic centimeters of serous fluid were withdrawn from the left pleural cavity. The following day, the respirations were less labored. The patient was given sulfathiazole for three days as a prophylactic against pneumonia and digitalis postoperatively.

She continued to improve steadily complaining less each day of pain. By July, 1941, she was allowed to sit up a little each day. The wound healed by primary intention. Her temperature, pulse, and respiration were normal. She was dismissed from the hospital on July 7, 1941, with no complaints.

COMMENT

Ligation of a patent ductus arteriosus has undoubtedly been established. Whether it will decrease the incidence of endocarditis is uncertain, especially in view of the postligation infected thrombus cited by Jones, Dolley, and Bullock.[21] In spite of that single case, there seems to be reason to believe that the operation may be successful in this respect. The use of the operation as a curative measure for patients with endocarditis and endarteritis must be regarded as highly experimental, but in view of the relentless course of the untreated disease, further operative undertakings are certainly in order.

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ANTERIOR DISLOCATION OF THE DISTAL EXTREMITY OF THE ULNA

Report of a Case

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AN ANTERIOR dislocation of the distal extremity of the ulna is undoubtedly a rare lesion, yet the distinctive mechanics of its production and the peculiar local pathologic changes make it an extremely interesting one. A report of the findings in a recently treated case was considered worth while.

CASE REPORT

A. A., colored female, aged 37 years. On Aug. 3, 1941, the patient was riding in a car which turned over. The dorsum of the forearm was on the ground, and the side of the car came down upon the flexor surface. Quite a bit of straining and twisting were necessary for the patient to disengage the arm. The patient had immediate pain and swelling at the wrist, with inability to use the wrist without increasing the local pain. She was brought to the hospital. X-rays were taken and reported as negative, hence the patient was told to apply hot soaks and to rest the extremity at home.

Three weeks later the patient returned to the hospital complaining of persistent throbbing pain in the right wrist. The pain became quite sharp with any attempted wrist motion or hand flexion. The pain was always referred to the volar aspect of the wrist. The swelling locally was most marked at this point but also extended distally to the finger tips and proximally to the midforearm.

On examination there was marked swelling of the whole distal third of the forearm, extending onto the hand. Point tenderness was localized over the volar aspect of the distal radioulnar joint. The distal extremity of the ulna was palpable in its dislocated position over the volar aspect of the wrist. Review of the x-ray films (Fig. 1*a* and *b*) taken at the date of original examination three weeks previously showed the anterior dislocation of the ulna with a fracture of the ulna styloid process. Motions at that time were

		RIGHT	LEFT
Wrist:			
	Flexion	15°	80°
	Extension	35°	90°
	Supination	From 15° to 80°	90°
	Pronation	0°	90°
Fingers:			
	Flexion	50%	100%
	Extension	90%	100%

On Aug. 23, 1941, an open reduction was done. The incision was made transversely across the wrist joint to the ulnar styloid and extended cephalad for a distance of approximately two inches (Fig. 2*a*). The skin flap was retracted, and

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dissection carried down along the edge of the flexor digitorum profundus, to the anterior aspect of the wrist joint. The distal extremity of the ulna was found lying upon the anterior aspect of the radius and displaced almost 1 cm. toward the radial side of the wrist. This displacement was firmly fixed by the contracted pronator quadratus muscle (Fig. 2*b*). The capsule of the wrist joint was opened transversely to expose this area for inspection. The triangular ligament and the detached ulnar styloid remained in their normal positions firmly attached to the

Fig. 1.—*a*. Typical displacement of ulna toward radial styloid process. *b*. Typical anterior displacement when true lateral x-ray projection is made. *c*. Anteroposterior view after reduction. *d*. Lateral view postreduction.

capsule, and could not be displaced when grasped with a forceps. No damage had occurred to the articular cartilage of the radius or ulna at the distal radioulnar joint. Following incision of the capsule and complete exposure of the radioulnar joint, a periosteal elevator was inserted as a lever and an attempt made to reduce forcibly the dislocated ulna. Even with strong force this could not be accomplished. The origin of the pronator quadratus was released from the lower one and one-half inches of the ulna, and immediately the dislocation could be reduced using an elevator

as a lever. Full pronation and supination could be demonstrated once this was done. The origin of the pronator quadratus was reattached and a capsulorraphy done to reinforce the support on the anterior aspect of the distal radioulnar joint.

The wrist and elbow were immobilized in a plaster of Paris splint maintaining the forearm in full supination, and held in this position for three weeks. Physiotherapy in the form of whirlpool baths, massage, and graduated exercise was continued for an additional three-week period.

At date of the last examination Nov. 21, 1941, the patient had resumed full activity as a housewife and maid, using the wrist and hand without disability, except for a sensation of slight stiffness in the wrist with full supination. Motions at this time were

Wrist:	Pronation	50°
	Supination	90°
	Extension	90°
	Flexion	45°

Full range of finger motion.

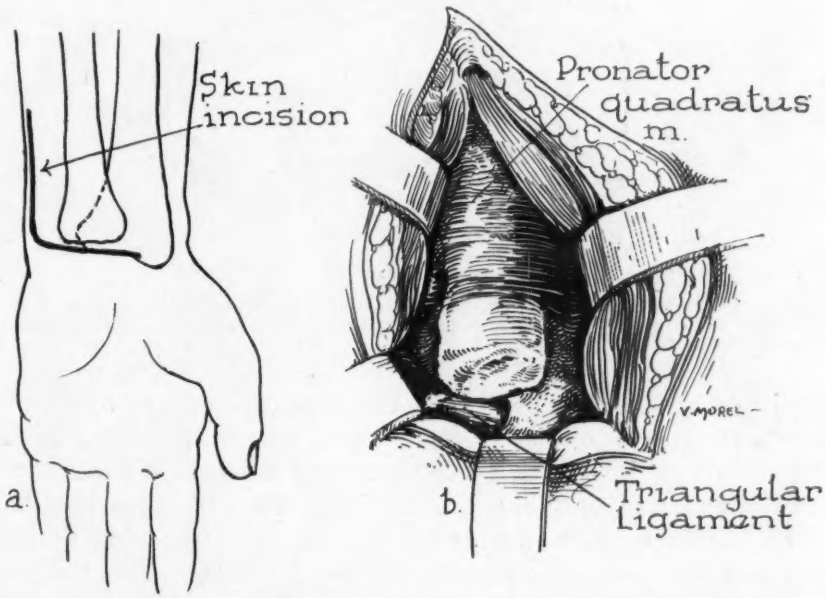

Fig. 2.—*a.* Outline of skin incision used. *b.* Drawing at operation showing deforming effect of pronator quadratus muscle, also intact undisplaced triangular ligament.

DISCUSSION

Uncomplicated anterior dislocation of the ulna was described for the first time by P. Desault in 1791 as an incidental finding on a cadaver. A complete description of the mechanics and a discussion of the immediate care were published by Cotton and Brickley in 1912[1] together with a résumé of 27 cases reported in the literature, in which résumé the exact mechanism of production of the dislocation is recorded. As a result of their investigations Cotton and Brickley felt that there were at least two mechanisms by which this lesion could be produced: (1) forced supination and (2) direct backward force on the hand with the ulna fixed mechanically or by force of triceps mechanism. There

is no evidence to dispute their conclusions. An interest in the lesion was aroused for a short time,[2-4] yet no description of the mechanics of the production of an anterior dislocation of the ulna, or of its aftercare has been included in any of the recent literature. In 1937 Vergoz and Choussat[5] reported sixty-eight cases compiled from the literature, but not including any of those reported in this country. Their report however is concerned with the reconstruction procedure used in the care of an old unreduced dislocation of the ulna.

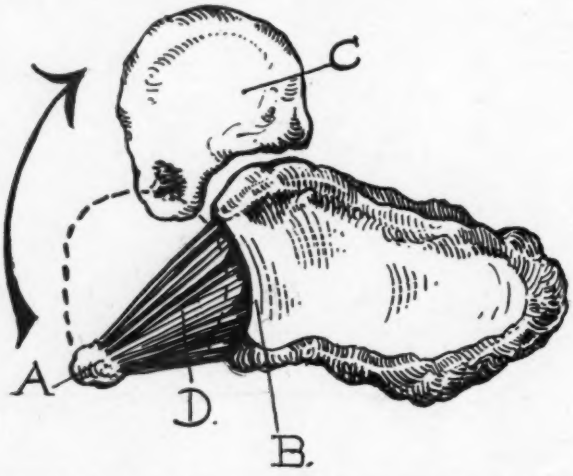

Fig. 3.—*A*. Styloid process of ulna. *B*. Attachment of triangular ligament to radius. *C*. Shaft of ulna displaced anteriorly and impinging upon volar aspect of radius. *D*. Triangular ligament.

Probably anterior dislocation of the ulna is always associated with either a fracture of the ulnar styloid or a tear of the triangular ligament at its point of insertion into the styloid process. When seen early before spasmodic contracture of the pronator quadratus muscle has occurred, reduction of an anterior dislocation of the ulna should be easily accomplished. The main force resisting reduction is the pull of the pronator quadratus. Where the interosseous membrane is not torn the displacement of the ulna may become mechanically fixed on the anterior aspect of the radius (Fig. 3). Traction against this force manually would be made much more effective with the administration of general anesthesia, or regional block anesthesia. In the more resistant cases, either skeletal traction exerted on the ulna itself or the use of a lever inserted between the radius and the ulna might be utilized with effect.

Where the dislocation has been allowed to remain for a period of time sufficient to allow cellular infiltration and fibrosis within the substance of the pronator quadratus muscle to occur, open reduction becomes necessary. Early simple open reduction followed by a capsulorraphy gives a satisfactory result. Later a more radical procedure must be done because of the organization which has developed in the distal

radioulnar joint. Sauvé and Kapandji[6] advise the production of a pseudoarthrosis in the ulna at the level of the distal third with replacement of the head of the ulna in its normal relationship with the radius, so as to allow free pronation and supination, yet prevent ulnar deviation of the hand at the wrist. Resection of the distal extremity of the ulna as advised by Darrach[3] would give a perfectly satisfactory result.

The disturbance in function of the flexors and extensors of the hand associated with this type of dislocation is sufficiently severe that emphasis must be laid upon the importance of reduction at the earliest possible moment.

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GROSS PATHOLOGIC ANATOMY OF THE UNUSUAL SHOULDER SPECIMENS IN TWO HUMAN CADAVERS, WITH SOME REMARKS RELATIVE TO THE SURGICAL SIGNIFICANCE OF THESE FINDINGS

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I. UNREDUCED ANTERIOR SUBCLAVICULAR DISLOCATION OF THE HEAD OF THE HUMERUS OF TWO YEARS' DURATION

THIS shoulder joint specimen is noteworthy because of the following features:

1. The anatomic relations of the dislocated humeral head, and the pathologic changes in the bones and soft tissues of the old and new articulations are defined.

2. The relative margin of safety to the adjacent vessels and nerves, and the functional integrity of the nearthrosis are emphasized.

3. The anatomic findings in this specimen of only two years' duration indicate that surgery of considerable magnitude would have been necessary to mobilize and reduce the dislocated humeral head, and that if reduction had been successful, in view of the advanced degenerative changes involving the humeral head and glenoid fossa, any procedure other than a resection of the humeral head or an arthrodesis of the shoulder joint might have proved inadequate. Indeed, the functional efficiency of the new shoulder joint, as was gathered from the history of the patient and as may be interpreted from the anatomic findings, might have made the wisdom of operative interference in this case questionable.

GROSS ANATOMIC FINDINGS

The specimen was obtained from the cadaver of a white female, aged 69 years, whose death had been due to a malignant lesion of the large intestine with metastases. She had sustained a traumatic dislocation of the right shoulder joint two years prior to her death, that had remained uncorrected after several attempts at closed reduction had failed.

Right Shoulder Region.—The undissected right upper extremity could be abducted at the shoulder to 45°, internally and externally rotated 45° each, and flexed and extended 45° each.

Muscles: The deltoid muscle was well developed and overlay the empty glenoid cavity. The coracobrachialis, short head of the biceps, and pectoralis minor muscles, arising from the coracoid process, stretched

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across the humeral head anteriorly. The teres major and pectoralis major muscles were attached to the margins of the bicipital groove and merged with the thick capsule enveloping the humeral head. The subscapularis lay below and anterior to the head and retained its insertion to the lesser tubercle. The long head of the triceps muscle was attached to the infraglenoidal tubercle. The combined tendons of the supraspinatus, infraspinatus and teres minor muscles (the musculotendinous cuff) overlay and terminated in the soft tissues filling the old glenoid cavity, having become completely detached from the greater tubercle.

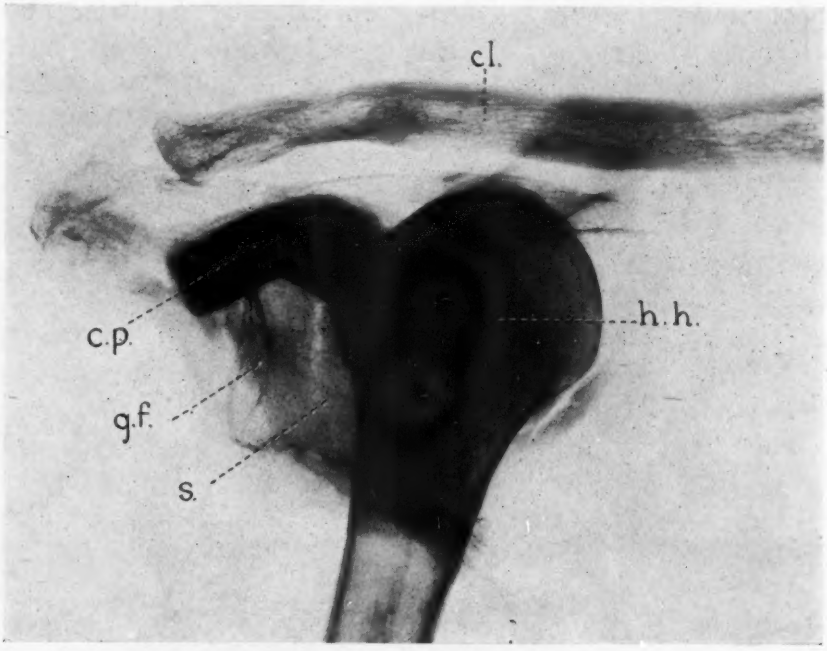

Fig. 1.—Anteroposterior roentgenographic view of the right shoulder region of Cadaver 1. The head of the humerus (*h.h.*) is displaced to a position anterior to the scapula (*s.*), medial to the glenoid fossa (*g.f.*) and the coracoid process (*c.p.*), and inferior to the middle third of the clavicle (*cl.*). There is marked decalcification of the bones. The articular surface of the humeral head is well defined, regular, and convex in its inner two-thirds, while its outer one-third is irregular and flattened.

Vessels and Nerves: The axillary vessels and brachial nerve plexus were deflected medialward and were angulated over the medial aspect of the humeral head. These structures were not visibly flattened or compressed and they presented no gross histologic changes in the region of the dislocated humeral head. The lumina of the vessels were patent, the arteries were well injected, and there were no varicosities. All of the muscles of the right shoulder, arm, forearm, and hand were as well developed as those on the left side, indicating the integrity of the nerve plexus during life.

The Nearthrosis: The humeral head was dislocated to a position anterior to the scapula, medial to the coracoid process and beneath the

middle third of the clavicle (Figs. 1 and 2). The head of the humerus was covered by a thick capsule, measuring 3 to 4 mm. in thickness, that was adherent to the anterior surface of the scapular neck and that was attached, laterally, to the medial margin of the hypertrophied coracoid process. The anterior, medial, and inferior portions of the capsule were inseparable from the overlying muscles. The capsule was opened by severing its coracoid attachment. Its inner surface was shiny and smooth and its cavity contained about 5 c.c. of yellow, mucoid fluid. It was somewhat fibrillated in the region of the lesser tubercle. The new joint cavity was completely closed and had no connection with the original glenoid cavity (Figs. 2 and 3). Microscopically, the lining of this new capsule was composed of one or more layers of flattened, modified connective tissue cells (Fig. 4).

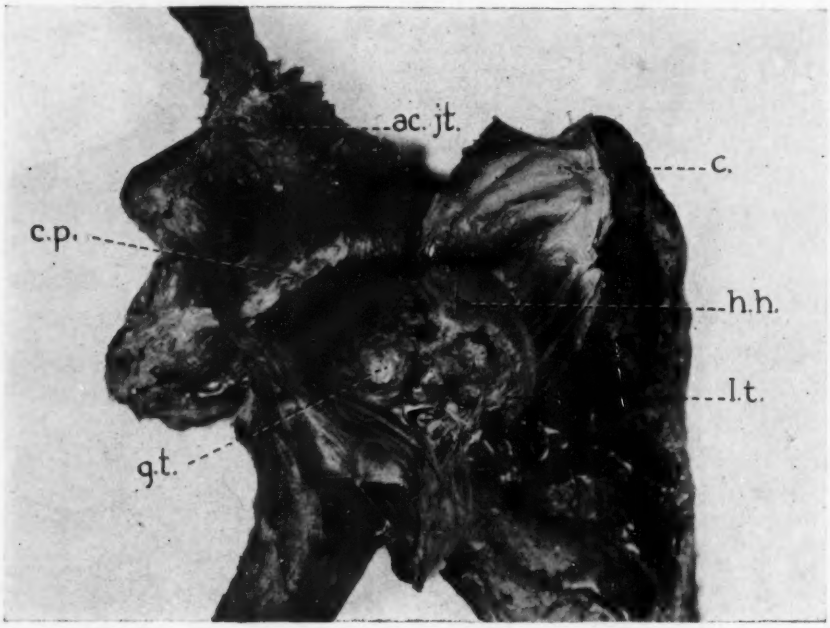

Fig. 2.—The right shoulder specimen of Cadaver 1, viewed anteriorly. The dislocated humeral head (*h.h.*) has been exposed by severing the overlying thick capsule (*c.*) at its attachment to the hypertrophied coracoid process (*c.p.*). This capsule has been reflected to expose its shiny articulating surface which is smooth throughout except for some fibrillation in the region of the lesser tubercle (*l.t.*). The cartilage over the medial two-thirds of the humeral head is fairly well preserved, although modified. It is absent over the flattened lateral one-third of the head of the humerus (that portion articulating with the coracoid process), with exposure of the underlying bone which is densified. The greater tubercle (*g.t.*) is smooth and without tendinous attachments, and the bicipital groove is shallow. The acromioclavicular joint (*ac.jt.*) shows advanced degeneration of its fibrocartilaginous tissues.

Coracoid Process: This was hypertrophied and presented on its medial aspect two facets, one posterosuperiorly and the other anteroinferiorly, that were smooth and eburnated and that articulated with the lateral portion of the humeral head (Figs. 2, 3 and 6).

Humeral Head: This was hypertrophied and approximately one-third larger than the head of the left humerus. Its medial two-thirds

was covered with thin, irregular and degenerated cartilage, while its lateral one-third was denuded of all cartilage with exposure of the eburnated subchondral bone. The humeral head was flattened considerably in its posterolateral portion and this region of the head articulated with an area on the anterior surface of the scapular neck, just medial to the glenoid fossa, that was elevated and relatively smooth, but the periphery of which was quite irregular, especially inferiorly and medially where the proliferative bone formed actual ledges. The thin posterior portion of the capsule was interposed between the humeral

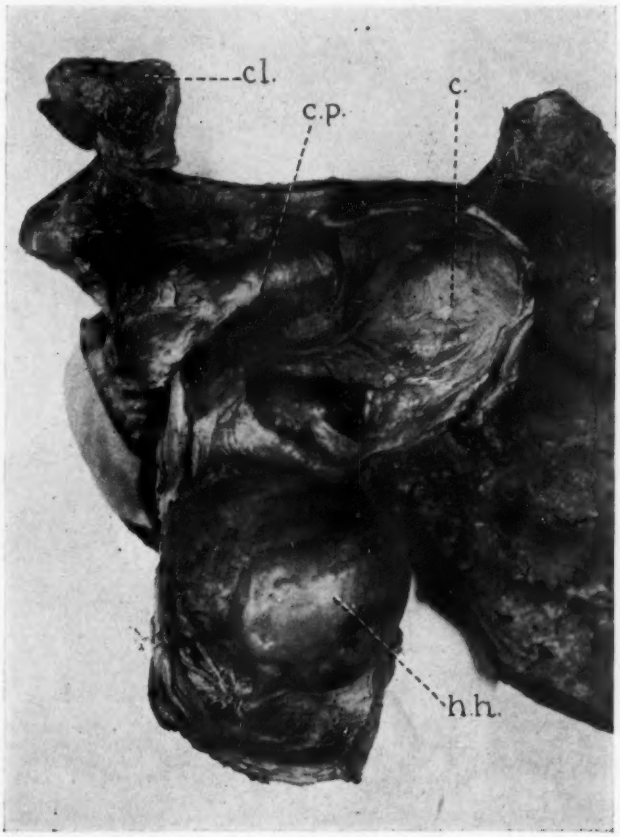

Fig. 3.—The right shoulder specimen of Cadaver 1, viewed from above. The humeral head (*h.h.*) has been rotated from its position in order to expose the new shoulder joint cavity. The internal surface of the thickened capsule (*c.*) of this closed cavity is smooth and shiny. The features of the humeral articular surface are well demonstrated from above. The medial two-thirds are covered with thin, modified hyaline cartilage, and the outer one-third (that portion articulating with the coracoid process *c.p.*) is rough, irregular, and denuded of cartilage with exposure of the subchondral bone. There is considerable flattening of the posterolateral portion of the humeral head, which area had articulated with the anterior surface of the scapular neck. The degenerated cartilage of the articular surface of the reflected clavicle (*cl.*) is demonstrated.

head and the modified anterior surface of the scapular neck. The greater tubercle was smooth and without tendinous attachments. The medial and lateral borders of the bicipital groove were less pronounced and the bicipital sulcus was more shallow in the right humerus than in the left humerus (Figs. 2, 3 and 6).

Glenoid Cavity: This was shallow and diminished in its antero-posterior dimensions, due possibly to fracture and displacement of the anterior lip of the fossa at the time of dislocation. The glenoidal labrum and the joint cartilage were absent, and the glenoid cavity was filled with soft tissue and the terminal portion of the musculotendinous cuff. The tendon of the long head of the biceps brachii muscle was intact and stretched from its attachment to the supraglenoidal tubercle, across the glenoid fossa to enter the bicipital groove about 4 cm. below the tubercles. The cavity of its sheath had no connection with the new shoulder joint cavity, but communicated at its posterior extremity through a small opening with the glenoid fossa (Fig. 5).

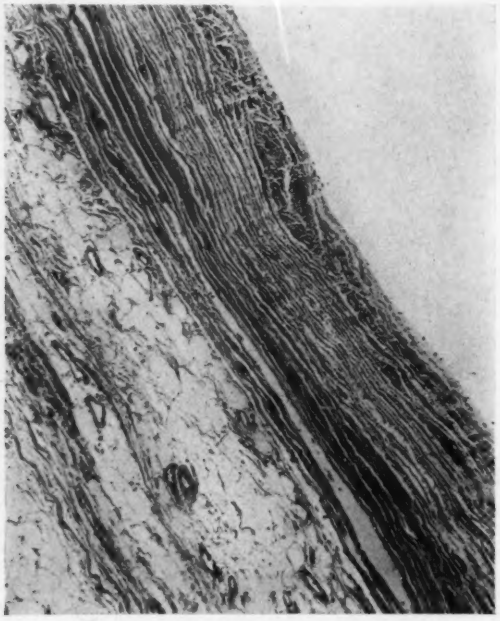

Fig. 4.—Photomicrograph (×50) of a section of the capsule from the new shoulder joint of Cadaver 1. The articulating surface is lined by flattened, modified connective tissue cells, supported by compacted connective tissue and muscle fibers.

Scapula: This was lighter in weight than the left scapula, despite the excessive proliferation of bone in the region of the glenoid cavity, acromion and coracoid processes, and scapular neck. The body of the scapula was thin and transparent. There was considerable atrophy of the cancellous and cortical bone of the head, neck, and shaft of the right humerus (Fig. 6).

Other Articulations.—Advanced degenerative changes were present in the left (undislocated) shoulder joint, both acromioclavicular joints, both knee joints, both elbow joints, and the spine.

Left Shoulder Joint: There was a complete circular defect (2 cm. in diameter) in the musculotendinous cuff, with marked fibrillation of the deep surface of the remaining portions of the cuff. The long bicipital

tendon was considerably frayed and degenerated and had become detached from the supraglenoidal tubercle. The cartilage of the glenoid fossa and humeral head was thinned and fibrillated, and in areas it was absent with exposure of the underlying subchondral bone. The margins of the glenoid cavity and the humeral head were irregular due to bony proliferative and regressive changes (Fig. 7).

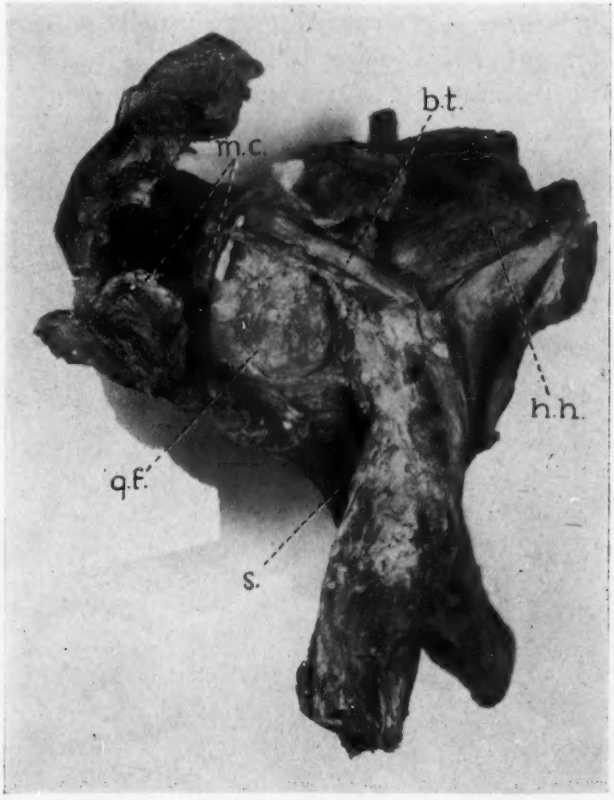

Fig. 5.—Lateral view of the right shoulder specimen of Cadaver 1. The long bicipital tendon (*b.t.*) is intact and extends, within its own sheath, from the supraglenoidal tubercle to enter the bicipital groove of the humerus about 4 cm. below the tubercles. The cavity of the sheath is closed except for a small posterior communication with the glenoid fossa (indicated by marker). The musculotendinous cuff (*m.c.*), which terminated in the soft tissues filling and overlying the glenoid fossa (*g.f.*), has been cut away to expose the contents of the latter. The position of the humeral head (*h.h.*) and shaft anterior to the scapula (*s.*) is well demonstrated.

DISCUSSION

Specimens of anterior dislocation of the humeral head have been described by Flower,[1] Broca and Hartman,[2] Delbet,[3] Pascalis and Monad,[4] Amabilis,[5] Petit-Dutaillis,[6] and Funck-Brentano.[7] These authors have stressed certain features which are well demonstrated in the specimen herein described: the flattened posterior portion of the enlarged head which articulates with the scapula and the inferior surface of the coracoid process; the formation of a new "glenoid fossa" on the anterior surface of the neck of the scapula; the new capsule enveloping the head,

that is formed, in part, from the deep surface of the subscapularis muscle, and that is reinforced, posteriorly, by the tendons of the short rotator muscles; the occasional ossified bodies within the tendons of the subscapularis and infraspinatus muscles, that articulate with the humeral head; the displaced tendon of the long head of the biceps muscle, and the contracted subscapularis muscle, both of which interfere with mobilization of the dislocated humeral head. In these descriptions, the axillary neurovascular structures were usually situated medial to the head of the humerus, although in the case of Funck-Brentano[7] they lay external to the head and within a gutter formed in a bony plaque

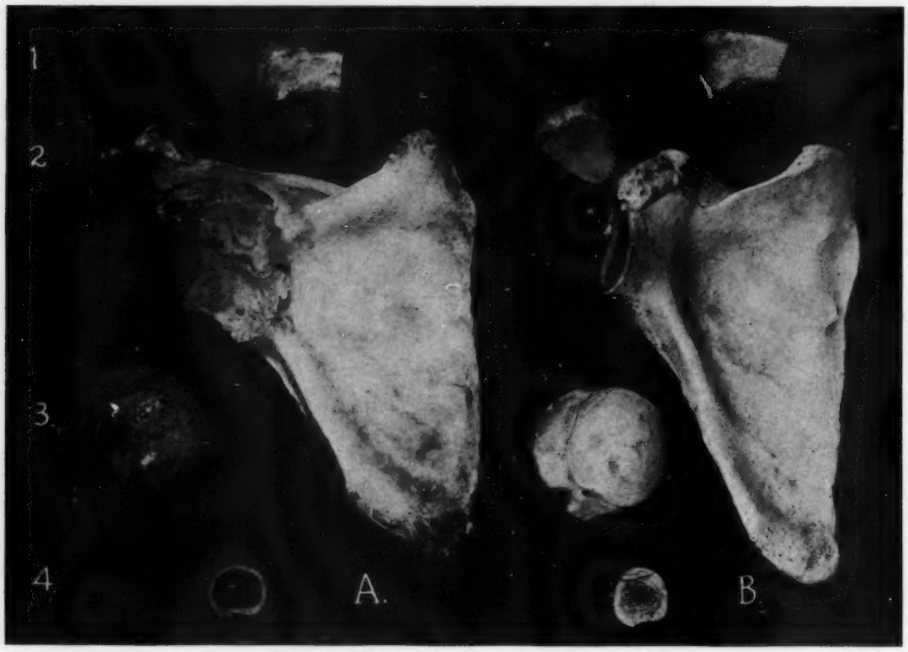

Fig. 6.—The macerated bones at *A* are those from the dislocated shoulder joint specimen of Cadaver 1, and those at *B* are for comparison and were removed from the grossly normal shoulder joint of a 44-year-old colored female.

1, Cephalic view of the acromial end of the clavicle; *2*, anterior view of the scapula; *3*, cephalic view of the humeral head; *4*, cephalic view of a transection of the shaft, 4 cm. below the anatomic neck.

A-1 demonstrates marginal proliferation at the acromial end of the clavicular segment. *A-2* demonstrates irregular bony proliferation involving the acromion process, the coracoid process, and the anterior surface of the scapular neck at the site of the new "glenoid fossa." There are two articular facets on the inferomedial surface of the hypertrophied coracoid process. The body of the scapula is thin and atrophic. *A-3* shows the flattening of the posterolateral surface of the dislocated humeral head, the irregularity of the subchondral bone, especially in the lateral one-third, and the regression of the greater and lesser tubercles. *A-4* demonstrates the advanced atrophy of the cortical and cancellous bone in the region of the humeral shaft, with relative enlargement of the marrow cavity.

within the subscapularis muscle. Atrophy of the deltoid muscle, indicative either of injury to the circumflex nerve or of disuse, was an occasional feature.

In some of these complete shoulder specimens and in many of the humeral heads resected by surgeons in the past for the correction of

recurring dislocation, a grooved defect, or, less often, a simple flattening was observed on the posterolateral surface of the humeral head (Hill and Sachs[8]). This "typical defect" has been observed shortly after a single dislocation and even prior to any attempts at reduction (Eve,[9] Hermodsson[10]), and for this reason it has been interpreted as representing a compression fracture of the spongiosa in this region of the humeral head, caused by the impingement of the posterolateral aspect of the head of

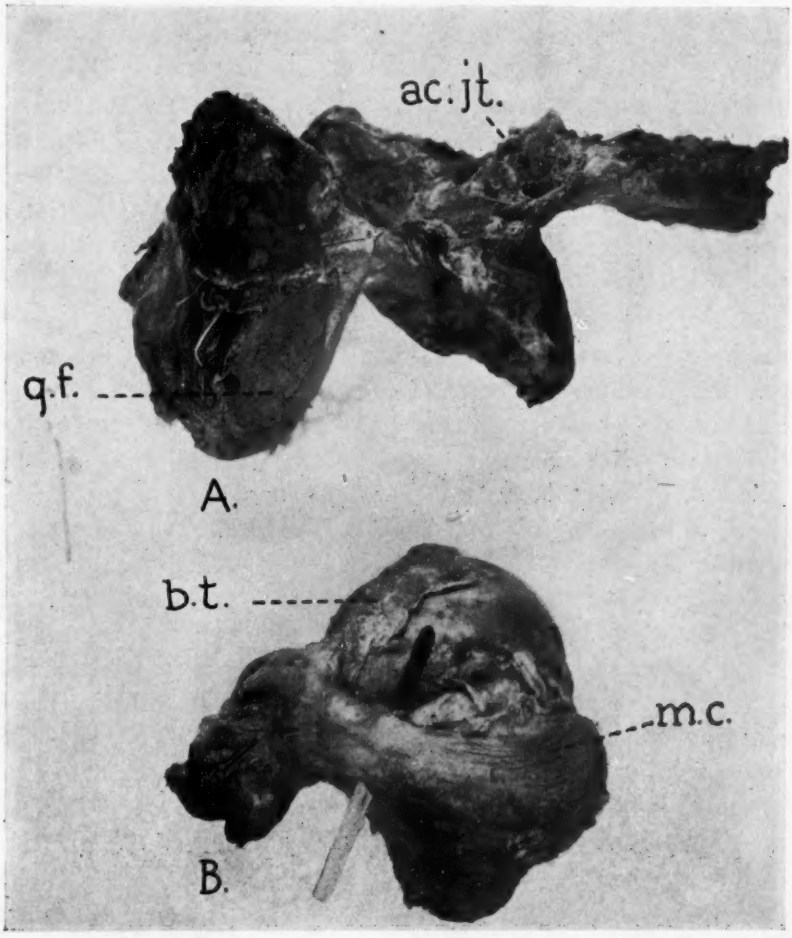

Fig. 7.—The left shoulder specimen of Cadaver 1. *A*, The glenoid fossa (*g.f.*) is covered with degenerated cartilage and demonstrates marginal bony proliferation. The articular surfaces of the acromioclavicular joint (*ac. jt.*) are the site of advanced degenerative changes. *B*, The musculotendinous cuff (*m.c.*) has been reflected over the humeral head and it demonstrates a large complete defect (indicated by the marker), and fibrillation of the deep surface. The capital portion of the long bicipital tendon (*b.t.*) is detached from the supraglenoidal tubercle, flattened, and moderately frayed.

the humerus on the anterior glenoidal rim. It has been suggested that this defect might be an etiologic factor in recurrent dislocation of the humeral head. Although no grooved defect was present on the posterolateral aspect of the head of the humerus in the dislocated specimen herein described, there was a distinct flattening in this region. While the

evidence was interpreted as favoring the belief that this flattening was attritional in origin, it cannot be denied that it might have represented an area of compression of the humeral head.

The pathologic anatomy in an *acute* anterior (subcoracoid) dislocation consists of the following—the humeral head passes through the anterior and inferior portion of the joint capsule at its glenoidal attachment, often carrying with it the anterior rim of the glenoidal labrum or the anterior lip of the bony fossa, and the margins of the capsular defect envelop the upper humeral shaft. The deltoid muscle and the musculotendinous cuff (short rotators) are stretched across the glenoid cavity and the latter may be avulsed at or near its bony attachments. The humeral head is displaced anterior to the scapula and deep to the subscapularis muscle. The tendon of the long head of the biceps muscle is displaced from the upper portion of the bicipital groove, with or without fracture of the greater tubercle, and it spans the glenoid fossa if it retains, as it often does, its attachment to the supraglenoidal tubercle (Key and Conwell[11]).

In *old* dislocations of the shoulder joint, as indicated in pathologic material and by the experience of those who have operated upon such cases (Codman,[12] Cubbins, Callahan, and Scuderi,[13] Meng and Miltner,[14] Bennett[15]), obstacles to closed or open reduction may be: the contracted and fibrosed subscapularis, pectoralis major and/or latissimus dorsi muscles; the tendon of the long head of the biceps muscle when it lies posterior to the head of the humerus and spans the glenoid cavity; the periarticular fibrous tissues (new capsule and its attachments) which bind the humeral head in its abnormal position; the torn musculotendinous cuff when it overlies and attaches to the soft tissues filling the glenoid fossa; the glenoid cavity, that becomes deformed by marginal bony proliferation and, in some instances, by loss of its anterior lip, and that loses its articular cartilage and becomes filled with scar tissue; the irregular proliferative bony process between the glenoid fossa and the articular surface on the neck of the scapula that receives the displaced humeral head; and the head of the humerus, that becomes hypertrophied and deformed and that loses its articular cartilage. Such changes in the bony, cartilaginous and synovial tissues may interfere with the reconstruction of a satisfactory shoulder joint even following the successful mobilization of the humeral head by division and release of the contracted tissues.

II. Advanced Degenerative Disease of the Shoulder Joint

These shoulder specimens from the same cadaver are noteworthy because of their following features:

1. They represent the most advanced stage of the degenerative processes that may involve this articulation.

2. The character of the degenerative lesions, such as the eburnated articular surfaces and the formation of several nearthroses, indicate that function may be preserved in the presence of advanced degenerative joint disease.

3. The ossified plaques in the deltoid muscle, bilaterally, which served as part of the receptive articular surface for the humeral head, represent bony metaplasia in the muscle and were probably produced by functional forces brought to bear by the humeral head after its erosion through the musculotendinous cuff. The shadow of these bony deposits on the roentgenogram could be misinterpreted for calcifications within the musculotendinous cuff or paratendinous soft tissues (Fig. 8).

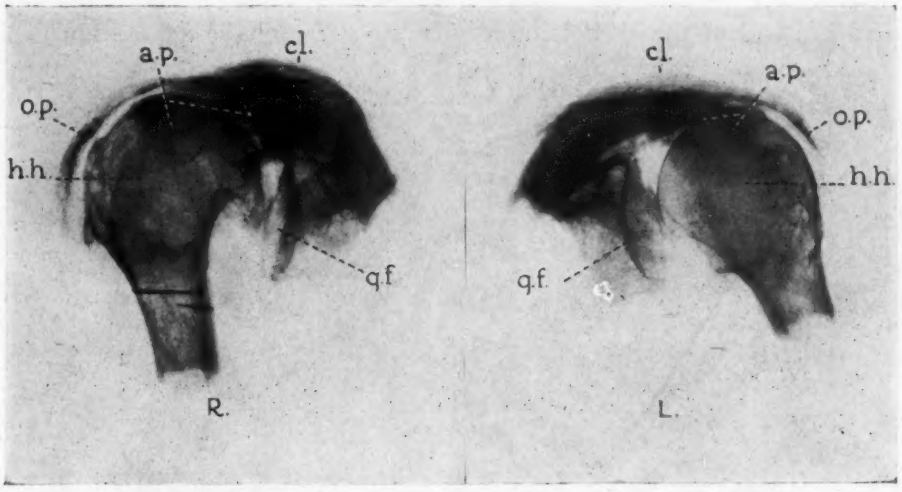

Fig. 8.—Anteroposterior roentgenographic view of the right and left shoulder specimens of Cadaver 2. In both specimens the humeral head (*h.h.*) articulates with a deep, acetabulum-like socket, made up of an ossified plaque (*o.p.*) within the deltoid muscle at its acromial attachment, the acromion process (*a.p.*), the acromial end of the clavicle (*cl.*), and the glenoid fossa (*g.f.*).

GROSS ANATOMIC FINDINGS

The specimens were obtained from a colored female, aged 77 years, who had died of bronchopneumonia.

Shoulder Joints.—The advanced degenerative and proliferative changes were similar in the right and left shoulder specimens, but were more marked on the right side.

The humeral head was observed to have eroded completely through the superior portion of the musculotendinous cuff of the shoulder (combined supraspinatus, infraspinatus, teres minor and subscapularis tendons), and only the peripheral attachments of this cuff and of the capsule remained. The humeral head articulated not only with the glenoid fossa but also with broad facets, formed by considerable bony proliferation, on the deep surface of the acromial end of the clavicle and on the deep surface of the acromion process, and with a bony plaque within the

substance of the thinned deltoid muscle at its origin from the acromion process. These four bony surfaces were separated from one another by narrow zones of fibrous tissue, yet their deep, concave surfaces presented one continuous, smooth articular surface for the reception of the humeral head, resembling very much an acetabulum. On the right side only, the normal interval between the glenoid fossa and the acromion process was completely filled with bone and this area was also smooth and eburnated. The cartilage of the glenoid cavity was fibrillated and thinned, and it was absent in the center of the fossa with exposure of the underlying bone. The glenoidal labrum was absent and replaced by marginal bony proliferation, especially anteriorly and posteriorly. The outer half of the humeral head (that is, that part articulating with the acromial end of the clavicle, acromion process, and the deltoid muscle)

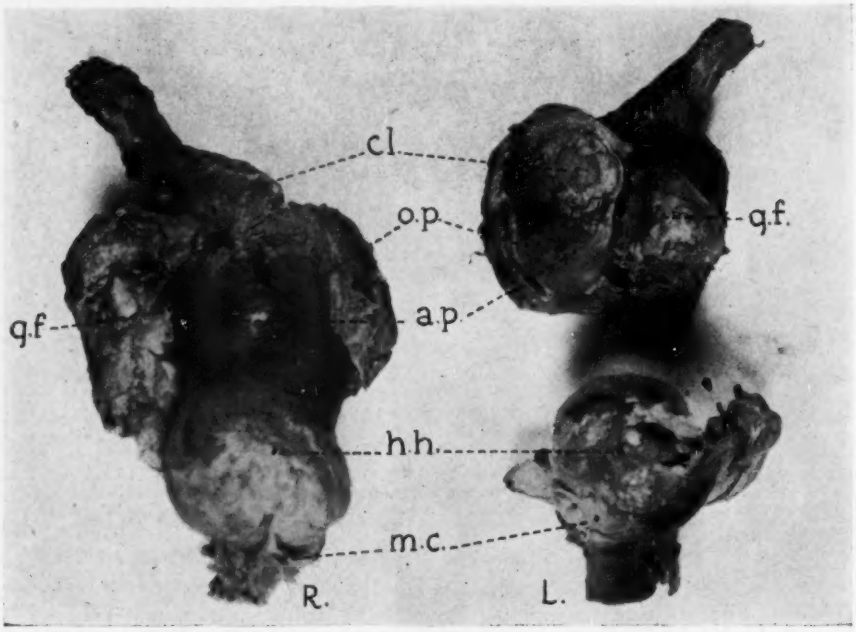

Fig. 9.—The right and left shoulder joints of Cadaver 2 have been opened in order to demonstrate the acetabulum-like sockets with which the degenerated articular surface of the humeral heads had articulated. This deep receptive surface, in both specimens, is composed of the glenoid fossa (*g.f.*), the eburnated undersurfaces of the acromial end of the clavicle (*cl.*) and of the acromion process (*a.p.*), and the ossified plaque (*o.p.*) within the deltoid muscle. Only the peripheral attachments of the musculotendinous cuff (*m.c.*) remain, the humeral head (*h.h.*) having eroded completely through the superior portion of the cuff, so as to articulate directly with the above bony and cartilaginous surfaces.

was flattened, eburnated in areas, smooth on the left side and very irregular on the right, and showed complete loss of its articular cartilage. The articular cartilage of the medial half of the humeral head (that is, that portion articulating with the glenoid fossa) was preserved but was considerably thinned and fibrillated, and it was absent in small areas with exposure of the subchondral bone. The humeral heads demon-

strated considerable bony proliferation, especially along their articular margins and in the region of the tubercles. This process was more advanced on the right humeral head where the bony proliferation had formed overhanging ledges (Figs. 8, 9, 10 and 11).

The synovia was thickened and in areas demonstrated villus and nodule formations. The deep surface of the deltoid muscle, adjacent to the bony plaque, was markedly fibrillated. The biceps tendon within

Fig. 10.—Macerated bony components of the right and left shoulder specimens of Cadaver 2. *1*, The inferior surface of the acromial end of the clavicle; *2*, the glenoid fossa (*g.f.*) and acromion process (*a.p.*), viewed laterally; *3*, the humeral head, viewed from above.

In each specimen, there is considerable bony proliferation about the articular facets on the undersurfaces of the acromial end of the clavicle and acromion process, and about the margins of the glenoid fossa. The interval between the acromion process and the superior glenoidal rim in the right specimen is filled with smooth, eburnated bone that presents an incomplete transverse fissure and that acts as part of the extensive articular surface for the reception of the right humeral head. The humeral heads, especially the right, demonstrate marginal bony proliferation and considerable irregularity of the articular surface.

the bicipital groove was frayed and flattened and it was attached to the bone at the proximal end of the bicipital sulcus, its capital portion being absent.

The oval-shaped plaques within the deltoid muscle measured 46 mm. in length, 22 mm. in width, and 4 mm. in thickness in the right shoulder specimen, and 26 mm. in length, 18 mm. in width, and 3 mm. in thickness in the left shoulder specimen. Their concave articulating surfaces were smooth and eburnated, while their superficial convex surfaces were irregular and merged with the fibrous and muscular elements of the deltoid (Fig. 11). Histologically, these plaques consisted of compact, adult lamellar bone (Fig. 12).

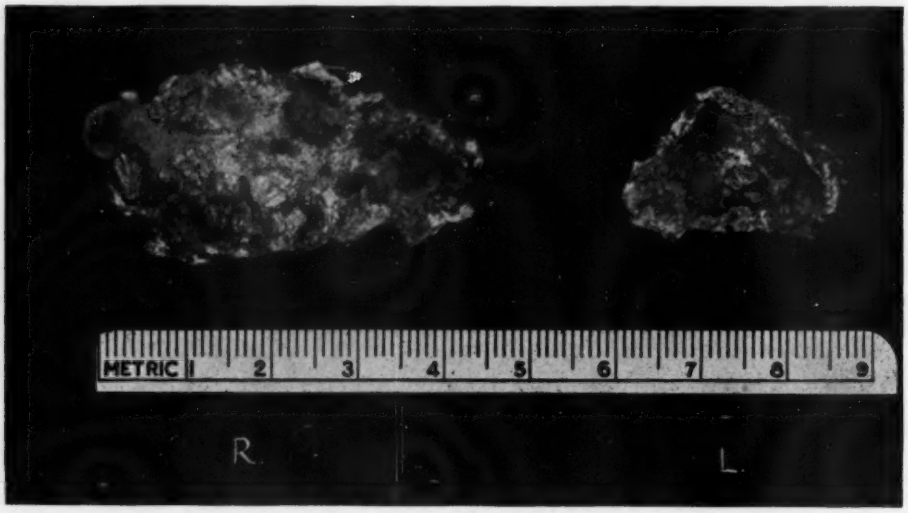

Fig. 11.—The oval-shaped bony plaques from the right and left shoulder specimens of Cadaver 2 are viewed from their deep, articulating side. These surfaces are concave, smooth, and eburnated.

Other Joints.—All of the other articulations in this cadaver showed degenerative changes in the synovia and capsule, and variable proliferative and regressive changes in the osseous structures. These lesions were very advanced in both knee joints, especially on the right side. They were moderately advanced in both acromioclavicular joints, both temporomandibular joints, the left elbow joint, both wrist joints, and in the small joints of the hands and feet, and these lesions were minimal, grossly, in both hip joints, both ankle joints, and the right elbow joint.

DISCUSSION

Degenerative joint disease is the result of wear and tear processes, of advanced age, and of repeated trauma which may be caused in a variety of ways. It has no relation to specific or nonspecific inflammatory processes. There is evidence to support Meyer's hypothesis of attrition,[16, 17] namely, that in addition to trauma and long-continued use

there is a failure of reparative processes to function adequately. A contributory factor of predisposition has been suggested, that is, that the type of cartilage inherited may be responsible for the earlier and more extensive changes encountered in some individuals (Bauer and Bennett[18]). That there may be constitutional factors, such as metabolic disorder or endocrine dysfunction, has been suggested but not proved. Generalized degenerative arthropathies are seen in such a metabolic pigmentary disorder as ochronosis. It has been demonstrated experimentally in laboratory animals that articular cartilage may become modified through the effects of excessive administration of certain hormones (Silberberg and Silberberg[19]), and it is possible that the deprivation of such hormones may also exert some deleterious effect on joint structure.

Fig. 12.—Histologic preparation (×50) of a segment of one of the ossified plaques in Fig. 11. This demonstrates adult lamellar bone architecture, with well-formed haversian systems.

The writer has examined most of the articulations of the upper and lower extremities and of the spine of forty-three cadavers, of which forty were male and two female, twenty-six white and seventeen colored, and with ages varying from 22 to 85 years. He has observed that, as a rule, the severity and extent of the degenerative lesions which involved the joints, grossly, were directly proportionate to the age of the subject. The joints involved by degenerative processes in the order of their frequency and severity were: (1) zygapophysial joints of the spine, (2) knee joints, (3) shoulder joints, (4) first metatarsophalangeal joints, (5) acromioclavicular joints, (6) elbow joints, (7) sternoclavicular joints, (8) temporomandibular joints, (9) wrist joints, (10) first metacarpophalangeal joints, (11) hip joints and (12) ankle joints. These

chronic functional lesions occur rather consistently in certain tissues and in certain areas of the same articulation. They have been investigated extensively by Meyer.[17]

In an examination of 335 shoulder specimens in 168 human cadavers, the following degenerative lesions have been observed by the writer: changes in the shoulder bursae, bicipital tendon sheath, and joint synovial membrane (thickening of the walls, fraying, villus and nodule formations, and adhesions); changes in the capsule and musculotendinous cuff (thinning, fraying, fibrillation, partial defects, and complete defects through the entire thickness of the capsule and cuff in 22 specimens or 6.6 per cent); changes in the tendon of the long head of the biceps muscle (flattening, fraying, fibrillation, dislocation and tear); degenerative changes in the articular cartilage of the humerus and glenoid fossa (fibrillation, thinning, denudation with exposure of the subchondral bone which may become polished and eburnated); and proliferative and regressive changes involving the humeral head, tubercles, bicipital groove and glenoid fossa. Meyer[16] has described more advanced stages in the progress of these degenerative processes in the shoulder region: fraying of the deep surface of the deltoid muscle, eventually with complete destruction of a large part of its midportion; partial or total destruction of the articular fibrocartilage and disks of the acromioclavicular joint, and erosion of the inferior capsule of this articulation, with or without joint polishing; formation of a true coracoclavicular articulation; and formation of a joint between the coracoid process and the humeral head and tubercles. The features of the shoulder specimen described in this communication, therefore, represent the degenerative joint lesion in its most advanced form.

In the series of shoulder specimens that I have studied, there was no certain instance, grossly, of calcification in the musculotendinous cuff, although it cannot be denied that roentgenographic and microscopic studies might have uncovered small deposits (Keyes[20]). Calcific deposits in the supraspinatus tendon were not observed by Sutro and Cohen[21] in a study of 100 shoulder joints in which changes incidental to senile degeneration were present in the supraspinatus tendon and contiguous structures. Such deposits, however, have been reported by Keyes[20] in two specimens in an examination of 192 shoulder joints and by Skinner[22] in two instances in a study of 100 shoulder specimens. One may conclude that calcification of the periarticular soft tissues is an uncommon feature in senile degenerative joint disease in the shoulder region.

These remarks are not to be confused with reference to those calcifications in the supraspinatus tendon and in the other short rotators of the shoulder joint that are found usually in younger subjects, not infrequently bilaterally, and that are asymptomatic until an acute inflam-

matory reaction is initiated by their extrusion into an adjacent bursa or tendon sheath. No adequate explanation has been offered to explain the origin of such calcific deposits. In an examination of the microscopic features of specimens removed surgically from thirteen such supraspinatus tendons with calcific deposits, variable degenerative and acute or chronic inflammatory processes were observed in the tendinous and paratendinous tissues. The areas of calcification were situated usually in the densely fibrous, poorly cellular and less vascular regions, and in no instance was there evidence of ossification in association with the calcified deposits. The failure of association of the latter two processes has been observed also in other forms of dystrophic calcification.

Codman[12] stated that he had found no instance of proved ossification of the supraspinatus tendon in his extensive anatomical and clinical material, and he stressed the need for distinguishing bits of tuberosities in the retracted tendons. Meyer[16] apparently also interpreted ossific nodules in the periarticular soft tissues as due to sudden trauma rather than to wear and tear. The ossified plaques in the shoulder specimens described herein, were composed of adult lamellar bone and probably represented bony metaplasia within the deltoid muscle. The humeral heads articulated in part with these areas of ossification, having eroded completely through the joint capsule and the musculotendinous cuff. In the absence of these bony plaques, the humeral heads might very well have eroded through the deltoid muscles and have come to lie subcutaneously. Such extensive stages of destruction must be very rare for they were observed by Meyer[16] only once in about one thousand bodies. That such ossific deposits may occur and that they will throw a shadow on the roentgenogram which is indistinguishable from that of calcifications in the periarticular soft tissues, should be appreciated by surgeons who are utilizing measures such as aspiration, irrigation, needling, or excision, for the removal of calcified deposits in the periarticular structures of the shoulder region.

CONCLUSIONS

1. A consideration of the anatomic and pathologic features in a cadaver specimen of an anterior subclavicular dislocation of the humeral head of two years' duration emphasizes the reasons for failure of attempts at closed reduction and for difficulty of open reduction of the displaced head of the humerus in such cases—the contractural changes in the muscles and other periarticular soft tissues, the interposition of bone and soft-tissue structures, and the modification of the articular components. Because of the marked degenerative changes which involve the bone, cartilage, and synovial tissues, a considerable problem presents itself, if the humeral head is successfully mobilized, in the reconstruction of the shoulder joint, in the restoration of its function,

and in the prevention of recurrence of the deformity. The recommendation of nonintervention, surgically, in selected cases of dislocation of the humeral head of long duration, where the functional integrity of the nearthrosis is moderately well preserved, is founded on an appreciation of these difficulties.

2. The shoulder specimens in a cadaver with degenerative lesions involving all of the articulations, demonstrate that the advanced stages of degenerative joint disease may be consistent with satisfactory articular function. In these specimens ossified plaques, formed by metaplasia in the deltoid muscle, contributed to the formation of a receptive articular surface for the degenerated humeral head. The shadows of these areas of ossification could be misinterpreted, roentgenographically, for calcifications within the musculotendinous cuff or paratendinous soft tissues of the shoulder region.

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THE EFFECT OF SYMPATHECTOMY ON RENAL BLOOD FLOW IN ESSENTIAL HYPERTENSION*

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THE mechanism by which certain operative procedures on the sympathetic nervous system produce clinical improvement in well-selected cases of essential hypertension is unknown. There are numerous theoretical possibilities which have been ably reviewed by Heinbecker[1] and Corcoran and Page.[2] One obvious explanation is based upon Smith's contention[3] that all cases of essential hypertension exhibit renal ischemia due to predominant constriction of the efferent glomerular arteriole; that is, that abolition of sympathetic impulses to the splanchnic region induces arteriolar relaxation and consequent increase in renal blood flow. If this is the correct explanation, the changes in the circulation pattern of the kidneys should be detectable by means of diodrast and inulin clearance studies. Alving, Adams, Grimson, Scott, and Sandiford,[4] Corcoran and Page,[2] and Selzer and Friedman[8] have reported no significant renal blood flow changes after operation in a few cases. It seems worth while to record our findings in five cases.

Our method of obtaining plasma clearances of diodrast and inulin as well as diodrast-Tm is being reported elsewhere;[5] it is essentially that devised by Smith.[6] Four of our patients were middle-aged women. Three of them, as well as the one male subject, aged 50 years, experienced entirely satisfactory symptomatic improvement, although in none was the mean blood pressure significantly reduced. One of the women received the supradiaphragmatic splanchnic section popularized by Peet on the other four patients the two-stage subdiaphragmatic operation advocated by Craig and Adson was performed. The one woman with a poor result belonged to this latter group. Preoperative clearance studies were done only after a prolonged period of hospital observation, when it was thought that the blood pressure had fallen as far as bed rest alone would permit. The studies were repeated at least ten days after operation when the patients were again comfortable and about to be allowed out of bed. In two instances a third set of observations was obtained about three months later. Unfortunately, diodrast-Tm was not determined preoperatively in two instances.

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Table I shows the pre- and postoperative data. Each figure is the average of at least three consecutive periods, and is expressed in terms of milligrams per 1.73 sq. M. surface area. DC and IC are the plasma clearances of diodrast and inulin, respectively. D-Tm is the maximal rate of tubular diodrast excretion in milligrams per minute. Whole blood flow data calculated from DC and hematocrit values have been omitted.

Our average normal values[5] are: DC, 544; IC, 117; D-Tm, 40; $\frac{IC}{DC}$, 0.205; $\frac{DC}{D\text{-}Tm}$, 13.56. All of these subjects, therefore, had considerable renal impairment, but none had azotemia. It should be pointed out that only E. A. had an abnormally high $\frac{IC}{DC}$ ratio and that she is the

TABLE I

PATIENT	DATE	DC	IC	D-Tm	IC/DC	DC/D-Tm	BLOOD PRESSURE
P. C., female, aged 50 years	11/14/40	329	63	31.2	0.19	10.54	175/110
	2/13/41	Supradiaphragmatic					
	3/ 7/41	333	59	25.5	0.18	13.05	185/125
	6/ 5/41	342	65	20.7	0.19	16.5	195/130
E. A., female, aged 45 years	1/29/41	180	56	26.6	0.31	6.76	205/120
	2/20/41	Second stage subdiaphragmatic					
	3/ 6/41	241	61	33.3	0.253	7.2	195/120
	6/ 6/41	237	66	36.6	0.28	6.47	180/110
A. G., female, aged 38 years	4/ 6/41	197	55	----	0.28	----	230/140
	5/10/41	Second stage subdiaphragmatic					
	5/21/41	185	50	14.2	0.26	13.0	250/160
H. B., male, aged 50 years	4/30/41	230	59	----	0.25	----	220/130
	5/13/41	Second stage subdiaphragmatic					
	5/22/41	289	68	23.7	0.235	12.2	200/110
S. P., female, aged 35 years	6/23/41	342	74	33.4	0.21	10.2	220/150
	6/30/41	Second stage subdiaphragmatic					
	7/10/41	393	81	47.5	0.20	8.3	210/140

only one whose rate of plasma flow per unit of tubular function, $\frac{DC}{D\text{-}Tm}$, fell clearly in the zone of renal ischemia. It was, therefore, probably unreasonable to anticipate postoperative renal hyperemia if ischemia could not be consistently demonstrated preoperatively, but it seems plain that even in this one instance no release of efferent arteriolar spasm took place. DC, IC, and D-Tm apparently increased proportionately after the operation, but the changes were not marked and we are inclined to disregard them.

In patient P. C., the increase in $\frac{DC}{D\text{-}Tm}$ tempts one to conclude that hyperemia was produced, but all $\frac{IC}{DC}$ ratios were well within normal limits, DC remained remarkably constant over a period of seven months,

and D-Tm fell progressively. To our knowledge, this phenomenon has not been described. It appears to represent progressive tubular degeneration.

Of the other three subjects there is little to be said except that no significant change in renal function occurred. It seems unlikely that inclusion of preoperative D-Tm in subjects A. G. and H. B. would have given cause to modify this statement.

DISCUSSION

We have elsewhere stated[5] that a high $\frac{IC}{DC}$ ratio is in itself incapable of demonstrating efferent glomerular arteriolar spasm because it can also be accounted for by diminished tubular extraction of diodrast. Smith[7] believes that renal ischemia is characterized by a low $\frac{DC}{D\text{-}Tm}$ ratio, and we have supported this contention. Subject P. C., however, shows how even this ratio may be altered by unexpected variations in tubular function, and adds one more complicating factor to the interpretation of clearances when no renal vein blood is available for renal extraction study. Diminished diodrast extraction may so alter DC and D-Tm values as to obscure coexisting renal ischemia.

There is nothing in this limited experience which leads us to believe that the circulation pattern of the human hypertensive kidney was appreciably altered by the surgical procedures employed. It might be argued that such an effect could be demonstrated only early in the disease before organic vascular changes take place, but it was precisely in this type of uncomplicated hypertension that we were unable to demonstrate consistent renal ischemia even by the $\frac{DC}{D\text{-}Tm}$ ratio.[5] It might also be argued that since none of these operations produced significant drops in blood pressure, no change in renal hemodynamics should have been expected. Having as yet no pertinent data, we are unable to answer this objection, but we believe that the changes in blood pressure which occasionally follow surgical attacks on the sympathetic nervous system are nonspecific in nature and not fundamentally related to the cause of the disease.

CONCLUSION

In five cases of essential hypertension, sympathectomy produced no important changes in renal blood flow.

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THORACOPLASTY THROUGH SHORT LATERAL AND POSTERIOR INCISIONS

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FOR some years we have tried to modify the usual incision for paravertebral thoracoplasty so as to cause less damage to muscles, with consequent opening of tissue planes, and to achieve a better cosmetic result. This last is of considerable importance in women, provided that the essential parts of the operative procedure can be properly performed with adequate rib removal and without undue trauma to tissues and the underlying lung.

First, we tried starting the usual posterior incision not higher than the level of the spine of the scapula, as first advocated by Roux, of Lausanne. This gives adequate exposure and saves the upper part of the trapezius and a small portion of the rhomboids. However, all the planes of the back have been opened, so that should a deep infection occur (fortunately a rare but most dreaded accident), the result is usually a complete dehiscence of the incision and a large, gaping, deep, grossly infected wound which at best will require a long, slow period of healing, often with resulting marked disfigurement. We next became interested in the multiple incision procedure proposed by Wangensteen and the muscle-splitting operation advocated by Head, but felt these were not entirely satisfactory. Hudson's transverse incision along the third rib anteriorly finally gave us the clue to what in our hands seems to be the solution to our problem. His incision, while perfectly suitable for men, is unsightly in women; nevertheless, it has demonstrated that the upper stage of a paravertebral thoracoplasty can be performed through some sort of anterior or lateral incision without any damage to the back muscles whatsoever. After working with Hudson's transverse anterior incision, a vertical anterolateral incision along the lateral border of the pectoralis major, and a vertical incision in the midaxilla, it becomes obvious that this last incision fulfills all the requirements: (1) Adequate exposure is obtained permitting the removal of the upper three or four ribs from the sternum to their necks if desired, either with or without the transverse processes; (2) the muscles and tissue planes of the back are not disturbed in any way; (3) no muscles are cut except the pectoralis minor, which may or may not be severed at its coracoid attachment, the insertions of the serratus magnus being stripped away

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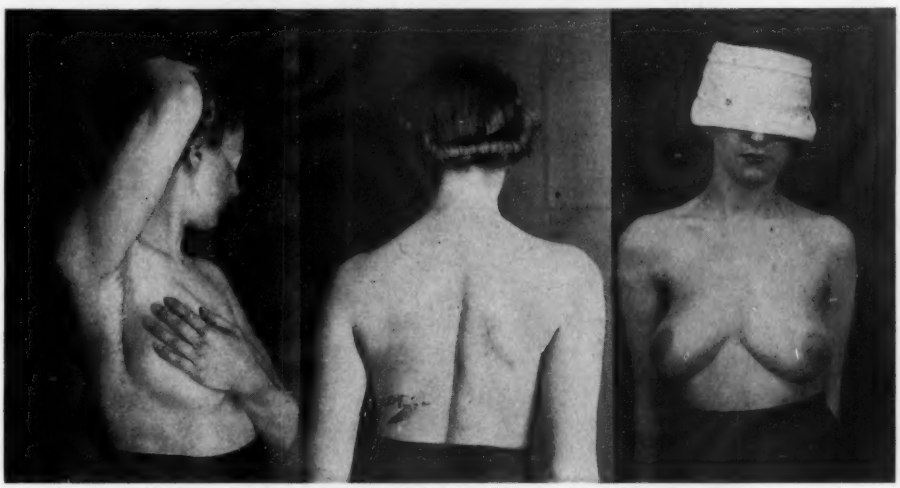
from the ribs along with the periosteum and still attached to it; (4) in case of deep infection only the skin and fascia of the incision need be reopened to drain the infected space adequately at its dependent point, thus insuring a more comfortable and shorter convalescence; (5) a fresh clean operative area for a lower thoracoplasty is maintained posteriorly; (6) a better cosmetic result is obtained; and (7) less bleeding and shock occur, making the procedure particularly useful in patients with poor reserve. Furthermore, if it becomes advisable to interrupt the operation because of unfavorable change in the patient's condition, the incision can be closed much more rapidly than can a posterior wound. We had hoped, also, that because of noninterference with the superficial back muscles there might be less tendency to scoliosis, but this has not proved to be the case. This incision is particularly desirable for patients with an empyema already drained posteriorly, as it enables one to remove the upper ribs and the anterolateral portions of the lower in a clean area away from the drainage opening.



A. *B.* *C.*

Fig. 1.—*A*, Axillary incision first stage; *B*, posterior incision second stage; *C*, anterior view of patient following completed thoracoplasty.

In employing this incision, instruments somewhat longer than is customary are highly desirable. Naturally, the exposure is not as wide as with the usual posterior approach, yet no undue difficulty should be experienced. Retracting is easy with abdominal and flat malleable retractors posteriorly and superiorly. Once the periosteum, the anterior scalene attachment, and the vessels are pushed away from the first rib, they and the lower trunks of the brachial plexus are readily held away from the rib with a long flat retractor while the posterior portion is cleaned to the tubercle or transverse process. The one disadvantage we have found has been that occasionally a true neuritis develops in the corresponding arm with even temporary weakness in the hand. We have

A.

B.

Fig. 2.—*A*, X-ray following first stage axillary incision; *B*, x-ray following completion of thoracoplasty by posterior stage.

attributed this to the rather prolonged stretching of the brachial plexus due to the position of the arm, and also to the pressure of the retractors. This has always cleared, however. Also, even with our long instruments we have felt the procedure to be less practical for excessively fat women or the occasional large, heavily musceled male.

The second and third stages of the thoracoplasty may readily be performed through a short incision around the angle of the scapula exposing the undefended intermuscular space between the trapezius and the latissimus dorsi muscles, the so-called auscultatory triangle. With the scapula in its relaxed position and after mobilizing the borders of the trapezius, the rhomboids, and the latissimus, the ribs may be readily removed from the third to the ninth or tenth. With scapula and muscles thus mobilized, the scapula is retracted about with great ease to expose any part of the operative field desired.

Both of these incisions are not new and have been in use in South America for some time. The Finochiettos have proposed these procedures and have extensively developed and advocated their use. Dr. Aguilar, also, is most enthusiastic about this method, and employs it routinely using local anesthesia throughout. His monograph on *Extrapleural Thoracoplasty* explains in great detail the development of the various incisions. We intend to present here only our experiences with them, with certain minor modifications.

PROCEDURE

Proper position of the patient is of the utmost importance. The patient is placed directly on his good side and the arm of the operated side elevated to an angle of about 120° with the trunk. The elbow is flexed to 90°, and the whole arm then carried forward across the body in order to relax the pectoralis major. To maintain this position, the forearm may be either bandaged or taped to the transverse bar of the ordinary ether screen frame. As is shown in the illustration, we have devised a much heavier frame constructed of strip metal. This fits into the lugs on the runners at the side of the operating table, and may be elevated or lowered as well as pushed forward or back. Since it is quite rigid, we have found it to be particularly serviceable whenever we have needed a support for the arm, as in this operation. In addition, the swiveled horizontal rods on either side of the transverse bar afford, when draped, a larger protected sterile field for the surgeon and his assistants.

After the skin has been prepared and the patient draped, a straight incision is made in the midaxilla between the pectoralis major and the latissimus dorsi. This begins at the lower border of the axillary hair and extends to the fifth rib, a distance of about five inches. The incision is carried through the axillary fat pad and the pectoralis major, identified and freed so that it may be lifted away from the chest. It is some-

times a good plan also to identify the pectoralis minor and divide it at its insertion into the coracoid, as this gives added relaxation and space as well as direct exposure of the thoracic cage. Next the long thoracic nerve is picked up and retracted, and the periosteum of the third or even the fourth rib incised through the fibers of the serratus from as far forward as desired to the posterior axillary line. This portion of the rib is stripped of periosteum and removed. The second rib is similarly treated except that it is divided somewhat farther back. The periosteum of the first rib, which is now found to be well exposed, is incised along the lower border from the cartilage to as far back as possible. With a protective finger along the upper edge of the rib, the periosteum is then stripped from the superior and inferior surfaces, and the attachment of the scalenus anticus separated with the long elevator. The vessels and

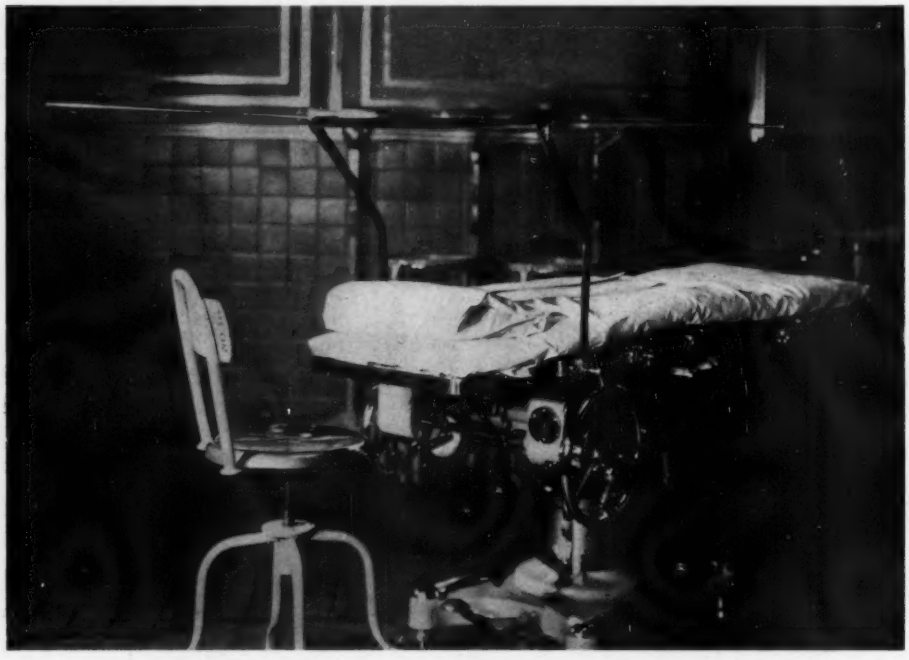


Fig. 3.—Anesthesia screen used for arm support showing movable wings.

brachial plexus are now gently retracted upward as the posterior portion of the rib is cleared. As the long ribbon retractor well protects these structures, the rib may be divided at or close to its tubercle with long rib shears and the remainder removed with a nine-inch Horsley rongeur. Once the first rib is removed the traction on the arm may be relaxed. The posterior ends of the second, third, and fourth ribs are now readily accessible. Starting with the second, the periosteum and muscles may be stripped back to and including the transverse process, and the rib and process removed with long rib shears and rongeurs. Instead, if preferred, the costovertebral joint may be entered with the long

concave spatula and the rib freed to its neck and disarticulated. The third rib is treated in like manner, and then the fourth if that also is to be removed. Ordinarily, except in cases of empyema, we advise the removal of only three ribs. Exceptions may be made, however, when it is felt that a four rib thoracoplasty is all that is required, or if there is no undue paradoxical respiration, the condition of the patient is excellent, and the type of lesion permits. After removal of the ribs, all that is necessary is to close the deep layer of the superficial fascia and skin, which we do with interrupted black silk sutures. There is very little bleeding throughout, little or no shock from the procedure, and so far in our experience a considerably more comfortable postoperative convalescence. The operation itself, however, is not a short cut, but takes time and patience with particular attention to position, exposure, relaxation, and adequate but gentle retraction. If the incision is carried too high into the axilla itself, a slight bowstring tendency may persist in the scar.

After the usual interval of about three weeks, the second stage is performed. As in the first stage, the patient is placed in the lateral position but with his uppermost arm at his side so as to relax the latissimus dorsi and thus the scapula. A short-curved incision about six inches long is made about the angle of the scapula to expose the intermuscular space. The presenting borders of the trapezius, rhomboids, and latissimus are picked up and the fascia dissected so as to mobilize them thoroughly. This provides perfect mobility of the scapula, which should be in its relaxed normal position at the start. With retractors under the scapula, and muscles, good exposure in any direction may be obtained, so that the desired number of ribs with or without transverse processes may be readily removed to as far forward as the midaxillary or anterior axillary line. Here again no muscles are divided, bleeding is minimal, and closure is attained by suture of only the superficial fascia and skin.

INSTRUMENTS

In order to facilitate removal of the ribs in both of the procedures just described, we have devised four instruments.* One of these is a slight modification of a periosteal raspatory and elevator† made some years ago for Dr. A. V. S. Lambert and one of us and consists of an Alexander type raspatory and a Doyen type curved elevator. For our present purposes we have lengthened the shaft of the raspatory end. This has not interfered with, and has actually enhanced the usefulness of the instrument for all other work on the thoracic cage. It is very strong and light, combines two instruments in one, and, due to its cylindrical body, fits comfortably into the hand without slipping or causing fatigue.

*With the kind cooperation of the Kny-Scherer Corporation.
†Made by Tiemann and Company.

The other three instruments were made particularly for this type of work. They form a set and are identical in weight and design. Each is ten inches in length and has a good-sized ovoid handle with several plane surfaces. This again permits of a comfortable grasp without slipping or cramping.

1. The shaft of the first is slightly decurved and ends in a raspatory of the Alexander type.

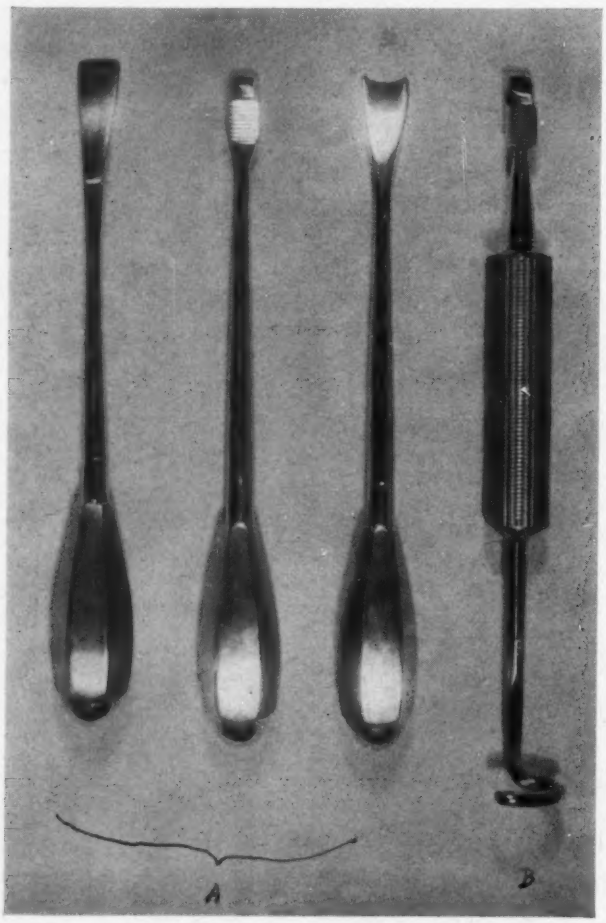


Fig. 4.—Special instruments described and advocated. *A*, Set of long shank elevators and raspatory; *B*, revised Berry-Lambert raspatory.

2. In the second instrument, the head consists of a slightly concave spatula, the center of which has a sharp cutting edge guarded on either side by the blunt slightly protruding margins of the head. 3. The head of the third is slightly concave and square with a straight drill edge and rounded dull corners. Although these instruments are not as long as those used by R. Finochietto and Aguilar, we have found them adequate. For cutting the ribs and transverse processes any of the long rib shears and rongeurs may be employed.

SUMMARY AND CONCLUSIONS

Two variations of the commonly used posterior hockey-stick incision for paravertebral thoracoplasty are described.

We believe these techniques to be the methods of choice in many instances because:

1. Both provide adequate exposure and permit thoroughness and safety of operation just as with the long posterior incision.
2. Neither one divides muscle, thus preserving the muscle and fascial planes of the back.
3. Only skin and fascia need be opened for drainage of a deep wound infection, which is usually localized by the barrier of the undisturbed muscles and fascial planes, thus avoiding the necessity of a wide gaping wound in the back.
4. By using the lateral incision for the first stage a fresh operative area is preserved posteriorly for the second stage.
5. Bleeding and shock are lessened in both procedures.
6. Convalescence is apparently more comfortable.
7. Better cosmetic results are obtained, an important consideration in women.

A disadvantage is the occasional temporary neuritis with weakness of forearm and hand that may develop after the first stage. This is probably due to stretching or retraction of the brachial plexus.

Special long instruments are described. These are not absolutely necessary, but instruments of equal or increased length greatly facilitate the procedure.

We wish to emphasize that similar operative techniques have been described and are being employed by Finochietto, Aguilar, and their associates. Their work undoubtedly preceded ours, but having arrived at the same conclusions quite independently, we believe it worth while to describe these modifications and along with them to urge their adoption in suitable instances.

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SHOCK IN CONTROL AND SPLENECTOMIZED ANIMALS UNDER ETHER AND PENTOBARBITAL SODIUM ANESTHESIA

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IT HAS been demonstrated by the work of Seeley, Essex, and Mann that sodium amytal (sodium iso-amylethyl barbiturate) alone or preliminary to ether anesthesia results in a marked delay in the onset of shock produced by intestinal manipulation as compared to results with ether anesthesia alone. Shock was considered to be present when the systolic blood pressure decreased to 70 mm. of mercury. With ether anesthesia alone shock occurred on an average of three hours and fifty-three minutes from the beginning of intestinal manipulation, while with sodium amytal alone shock did not appear on the average until eleven hours and thirty-three minutes from the time intestinal manipulation was begun. One of us (Kendrick) obtained strikingly similar results with ether and pentobarbital sodium using the methods described by Seeley, Essex, and Mann. Because of the possibility of a practical application of these findings to the care of patients injured in war or in accidents, further work on this problem seemed advisable.

As a result of a series of researches in this laboratory (Searles and Essex; Essex, Seeley, Higgins, and Mann; and Hausner, Essex, and Mann) it has also been demonstrated that anesthesia with certain barbiturates causes hemodilution for which the spleen is largely responsible since this organ under the influence of barbiturates removes from currency a large volume of erythrocytes. We wished to ascertain whether the spleen plays a role in the delay in the onset of shock when barbiturates are used. It may logically be asked whether the barbiturates would be of value in delaying the onset of traumatic shock induced under ether anesthesia if given to control and splenectomized dogs when shock was considered imminent. If so, when would be the optimal time to give the drug and how long after intestinal trauma might it be expected to produce effective results? The work described in this paper was done in the hope of answering these questions.

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Work done at the Institute of Experimental Medicine.
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METHODS

All experiments were done on dogs using ether and pentobarbital sodium anesthesia. Shock was produced by delivering the small intestine outside of the abdominal cavity and gently massaging it throughout its entire length for thirty to forty minutes, after which it remained exposed on the anterior abdominal wall. The blood pressure in the femoral artery was recorded on a standard kymograph. As in previous experiments in this laboratory the animals were considered to be in a state of shock when the systolic blood pressure had declined to 70 mm. of mercury. It was recognized that the arterial blood pressure might not accurately reflect the physiologic state of the animal. For that reason the blood pressure was not used as the criterion for the proper time to administer pentobarbital sodium. For this purpose the degree of hemoconcentration as determined by a Cenco-Sheard-Sanford photelometer was considered more satisfactory. It had been found in the studies to which reference has already been made that the animals were in shock as indicated by the blood pressure when the average concentration of hemoglobin was 130 to 135 per cent, the preanesthetic concentration being arbitrarily called 100 per cent. Thus when the hemoconcentration had reached 130 to 135 per cent shock was considered imminent even though the blood pressure was being maintained near the control level.

The animals were anesthetized initially with ether, and narcosis was maintained by autoinhalation.[2] After the desired hemoconcentration had been attained ether anesthesia was discontinued and pentobarbital sodium was given intravenously in doses of 10 to 15 mg. per kilogram. In three experiments pentobarbital sodium was given when the hemoconcentration had reached 120 per cent, while in four experiments the hemoglobin was allowed to concentrate as much as 135 to 140 per cent before pentobarbital sodium was injected. To avoid a marked depression of blood pressure the drug was given very slowly in all experiments. In the series of seven experiments on splenectomized animals the spleen was removed immediately preceding intestinal manipulation.

RESULTS, SPLEEN PRESENT

In the four experiments in which the hemoglobin was allowed to concentrate 135 to 140 per cent, it required an average of twelve hours and thirty-five minutes to reduce the systolic blood pressure to 70 mm. of mercury. The average time required for the hemoconcentration to reach 130 to 140 per cent was three hours and eleven minutes. The average time that elapsed from the beginning of intestinal manipulation until the administration of pentobarbital sodium (15 mg. per kilogram) was three hours and fifty minutes. In the three experiments in which pentobarbital sodium was given when the hemoglobin had concentrated

to only 120 per cent, the systolic blood pressure did not decrease to 70 mm. of mercury until thirteen hours and twenty minutes after intestinal manipulation was begun. The greater the percentage of hemoglobin concentration at the time pentobarbital sodium was given the more rapidly shock occurred.

Rapid concentration of the blood always occurred while the animals were under ether anesthesia. When the administration of ether was discontinued and after pentobarbital sodium was given, a decrease in hemoglobin invariably occurred but hemoconcentration slowly developed as the experiments were continued (Fig. 1).

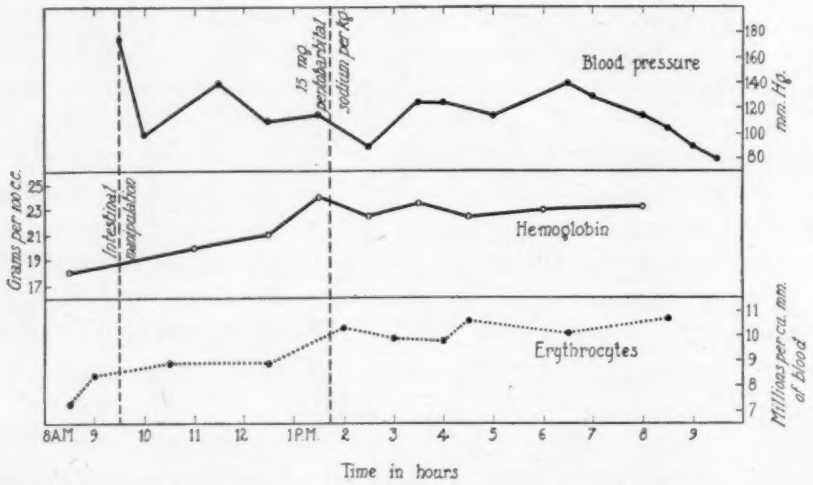

Fig. 1.—The effect of ether anesthesia, intestinal manipulation, and an injection of pentobarbital sodium on the blood pressure, hemoglobin concentration, and the number of erythrocytes in a dog with the spleen present.

RESULTS, SPLEEN ABSENT

In five experiments on splenectomized animals pentobarbital sodium in doses of 15 mg. per kilogram of body weight was injected intravenously after the hemoglobin had concentrated 135 to 140 per cent. The average time required for the systolic blood pressure to decline to 70 mm. of mercury was eight hours. During ether anesthesia the hemoconcentration was rapid. Following the injection of pentobarbital sodium the hemoconcentration increased, but much more slowly. The decrease in hemoglobin observed in experiments in which the spleen was present was not seen in the present series (Fig. 2).

In order to determine the effectiveness of smaller doses of pentobarbital sodium two experiments were done in which 10 mg. of the drug per kilogram of body weight were given after the hemoglobin had concentrated 130 to 140 per cent. In one experiment the blood pressure declined to shock level in eleven hours and forty-five minutes and in the other experiment shock occurred eleven hours and thirty minutes after intestinal manipulation.

COMMENT

The results of the experiments just reported indicate that doses of pentobarbital sodium as small as 10 mg. per kilogram of body weight were effective in delaying the decline of the blood pressure to a shock level even though intestinal manipulation and ether anesthesia had caused a concentration of hemoglobin that indicated that shock was impending.

That the spleen is an important factor in causing an apparent hemodilution following the injection of pentobarbital sodium has again been demonstrated by experiments on splenectomized animals in which hemodilution was not observed. The fact that the blood pressure was more rapidly reduced to a shock level in the splenectomized than in the nonsplenectomized dogs would seem to indicate that the spleen is of considerable aid to the organism in its resistance to shock. The reservoir function of the spleen was probably its chief contribution. In the absence of the spleen the injection of pentobarbital sodium caused delay in the decline of blood pressure to a shock level as compared to results with ether anesthesia alone. The mechanism by which pentobarbital sodium is effective in this respect is not known. However, it should be emphasized that the animals in which the onset of shock was longest delayed were those that had received the smallest doses of pentobarbital sodium.

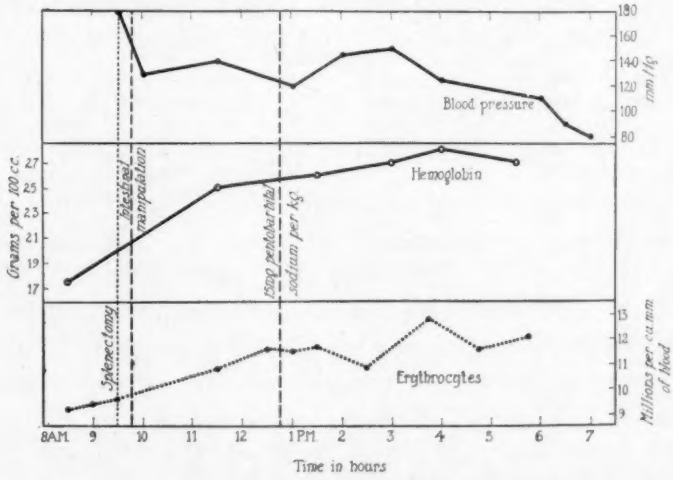

Fig. 2.—The effect of ether anesthesia, intestinal manipulation, and an injection of pentobarbital sodium on the blood pressure, the hemoglobin concentration, and the number of erythrocytes in a dog splenectomized just before the intestine was manipulated.

SUMMARY AND CONCLUSIONS

In this study an effort was made to determine the effectiveness of pentobarbital sodium in delaying shock when given after the hemoglobin had concentrated to a point at which shock might be considered

imminent. The small intestine of dogs was manipulated under ether anesthesia. When the hemoconcentration had reached the desired level (120 to 140 per cent) pentobarbital sodium was given and ether anesthesia was discontinued. When the systolic blood pressure reached 70 mm. of mercury the animals were considered to be in a state of shock. A series of experiments was done on both nonsplenectomized and splenectomized dogs. In the former experiments a decrease in hemoglobin occurred immediately following the injection of the drug but this was followed by hemoconcentration which developed slowly. In the absence of the spleen hemodilution did not occur. In every experiment the greater the percentage of hemoglobin concentration the more rapidly shock developed. Under the conditions of our experiments the splenectomized animals went into shock more rapidly than the nonsplenectomized series of animals, which may be taken as an indication that the spleen is a definite aid to the organism in its resistance to shock. In all experiments, whether or not the spleen was present, shock developed more slowly than in previous experiments in which ether alone was used.

The evidence appears conclusive that shock develops more slowly in animals under pentobarbital sodium than in animals under ether anesthesia. The reason for this result is not known. These findings should not be taken to mean that the administration of pentobarbital sodium may be used as a prophylactic procedure in the treatment of the injured but rather that this drug is indicated as a supportive agent if the injured are to be given a general anesthetic such as ether.

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SOME OBSERVATIONS ON THE RATE OF ABSORPTION FROM VARIOUS BODY TISSUES

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DURING the course of recent investigations concerning reactions to procaine in experimental animals, it appeared evident that the rate of absorption differed greatly according to the route of administration.[1] Since these experiments suggested that death ensued when the blood concentration of the drug reached a certain critical level, regardless of the site into which the toxic dose of procaine was injected, and since the circulating procaine is known to be rapidly destroyed or detoxified, it was felt that the wide differences in the lethal dose with different routes of administration were a reflection of different rates of absorption into the blood stream. These considerations led me to devise some simple experiments which might throw additional light upon this problem.

In all of the experiments except those dealing with the absorption of sulfanilamide, some indirect end point was selected rather than frequent and repeated chemical analysis of the blood which would have rendered the problem much more difficult. The general agreement of the experimental results obtained by using different effects of the various drugs which might reflect a therapeutic, toxic, or lethal blood concentration of the injected drug indicates that the method is valid. In all the experiments absorption from the subcutaneous, intramuscular, intrapleural, intraperitoneal, and intravenous routes of administration was studied. With each route control studies with distilled water given in the maximal amounts employed in the case of the drugs were carried out, and no symptoms and no deleterious effects were noted. Following intrapleural injections the animals were examined at autopsy and those whose lungs had been inadvertently damaged were excluded from consideration.

EXPERIMENTAL RESULTS

In all the experiments there seemed to be most rapid absorption from the intrapleural route. With some drugs, peritoneal absorption was apparently slower than that from subcutaneous or intramuscular tissues; with others it was more rapid.

Procaine.—In Table I are listed the results obtained from a large number of guinea-pigs given varying amounts of procaine hydrochloride by different routes. The median lethal dose varied from 41 mg. per

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TABLE I

SHOWING VARIATION IN MEDIAN LETHAL DOSE OF PROCAINE IN GUINEA-PIGS ACCORDING TO THE ROUTE OF ADMINISTRATION

ROUTE OF ADMINISTRATION	MEDIAN LETHAL DOSE IN MG. PER KG.	NUMBER OF ANIMALS USED
Intravenous	41	54
Intrapleural	42	25
Thoracic paravertebral	115	10
Lumbar paravertebral	250	30
Intramuscular	330	25
Subcutaneous	410	56
Intraperitoneal	750	28

TABLE II

TIME ELAPSING (IN SECONDS) AFTER INJECTION OF STRYCHNINE INTO GUINEA-PIGS BEFORE ONSET OF CONVULSIONS. LETHAL EFFECT OF VARYING DOSES ACCORDING TO ROUTE OF ADMINISTRATION

DOSE IN MG. PER KG.	ROUTE OF ADMINISTRATION				
	INTRAVENOUS	INTRAPLEURAL	INTRAMUSCULAR	SUBCUTANEOUS	INTRAPERITONEAL
0.16	No			No	
0.48	7	No		No	
0.64		8		No	No
1.6	5 D	20(2) D	No	No	No
2.4	3.5(3) D	16(3) D	215(3) D*	No(3)	No(3)
3.84	2.5 D	11 D	140 D	540 D	No
5.76	1.5 D	15 D	132 D	360 D	No
9.6		6 D	118 D	340 D	420 D

Absence of convulsions is indicated by "No."
When more than one animal was used, the number is put in parentheses.
"D" indicates that death occurred.
(These designations are also used in Tables V, VI, VII, VIII, and IX.)
*Two of the three animals died.

TABLE III

TIME ELAPSING (IN SECONDS) AFTER INJECTION OF STRYCHNINE INTO MICE BEFORE ONSET OF CONVULSIONS. LETHAL EFFECT OF VARYING DOSES ACCORDING TO ROUTE OF ADMINISTRATION

DOSE IN MG. PER ANIMAL	ROUTE OF ADMINISTRATION				
	INTRAVENOUS	INTRAPLEURAL	INTRAMUSCULAR	SUBCUTANEOUS	INTRAPERITONEAL
0.02	5 (5) D	25(7) D	No	No	No
0.04	1.7(3) D	23(3) D	250(3) D	370(3) D*	D†

*Two of the three animals died.
†Only one of the four animals had convulsions and only one died.
The mice weighed between 25 and 30 Gm.

TABLE IV

TIME ELAPSING (IN SECONDS) AFTER INJECTION OF MORPHINE SULFATE INTO MICE BEFORE ONSET OF SLEEP AND BEFORE DEATH

DOSE IN MG. PER ANIMAL	ROUTE OF ADMINISTRATION									
	INTRAVENOUS		INTRAPLEURAL		INTRAMUSCULAR		SUBCUTANEOUS		INTRAPERITONEAL	
	SLEEP	DEATH	SLEEP	DEATH	SLEEP	DEATH	SLEEP	DEATH	SLEEP	DEATH
155*	30(2)	80(2)	320(2)	450(2)	No	No	2400	2700	No	No
167†	1	25	297(3)	465(3)	600	3900	4200	4400	1100	No

*Also 4 mg. atropine.
†Also 4.5 mg. atropine.

kilogram with intravenous injections to 750 with intraperitoneal injections. The median lethal dose was very small with intravenous and intrapleural injections and was progressively larger when the drug was given in the thoracic paravertebral space, lumbar paravertebral space, intramuscular tissues, subcutaneous tissues, and the peritoneal cavity. Apparently the rapidity of absorption of the drug varied in this order.

Strychnine.—Strychnine was administered in different doses and by various routes to guinea-pigs and to mice. The results are recorded in Tables II and III. The interval after injection of the drug before the onset of convulsions was shortest when the drug was given intravenously. It was brief following intrapleural injections, somewhat longer after intramuscular injections, still longer after subcutaneous injections, and longest after intraperitoneal injections. Also, it will be noted that the rapidity or the delay in onset of convulsions varied directly with the size of the lethal dose. One would judge that the rate of absorption of the drug is most rapid from the pleura, somewhat slower from the muscles, still slower from the subcutaneous tissues, and slowest from the peritoneum.

Morphine.—The same conclusions are reached from study of the interval following injection of morphine into mice before onset of sleep and of death (Table IV).

Phenolsulfonphthalein.—At intervals of a week a normal, healthy mongrel dog was given 1 c.c. of phenolsulfonphthalein by various routes following introduction of a catheter into the bladder. The appearance time of the drug in the urine was noted, total specimens were collected at fifteen-minute intervals, and the excretion curve was plotted. An intravenous injection was carried out at the beginning and at the end of the experiment. The agreement in these excretion curves was evidence that no essential change in kidney function had occurred. The intravenous excretion curve in Fig. 1 is an average of the two. If the rapidity of excretion is a rough measure of the rapidity of absorption, one would conclude that absorption is most rapid from the pleura, less rapid from the subcutaneous tissues, and slowest from the peritoneum.

Metrazol.—Different amounts of metrazol were administered by various routes to guinea-pigs and to mice, and the interval after injection before onset of convulsions was recorded (Tables V and VI). Judging by these observations and by the lethal dose with the different routes, one would assume that the drug is absorbed most rapidly from the pleura, less rapidly from the peritoneum, still more slowly from the muscles, and most slowly from the subcutaneous tissues.

Coramine.—Similar experiments were carried out with coramine (Tables VII and VIII) and similar results were obtained.

Nembutal.—A number of mice were given 2.5 mg. of nembutal by several routes, and the interval before sleep was noted. The onset of sleep was almost instantaneous after intravenous injections, was very

TABLE V

TIME ELAPSING (IN SECONDS) AFTER INJECTION OF METRAZOL INTO GUINEA-PIGS BEFORE ONSET OF CONVULSIONS. LETHAL EFFECT OF VARYING DOSES ACCORDING TO ROUTE OF ADMINISTRATION

DOSE IN MG. PER KG.	ROUTE OF ADMINISTRATION				
	INTRAVENOUS	INTRAPLEURAL	INTRAPERITONEAL	INTRAMUSCULAR	SUBCUTANEOUS
0.2	4				
0.3	4 D	11	No	No	No
0.4	4 D	11 D	180	230 D	720
0.6	1.5 D	6.5(2) D	120	540	1800
1.0	1.5(2) D	9 D	100 D	100 D	600 D

TABLE VI

TIME ELAPSING (IN SECONDS) AFTER INJECTION OF METRAZOL INTO MICE BEFORE ONSET OF CONVULSIONS

DOSE IN MG. PER ANIMAL	ROUTE OF ADMINISTRATION				
	INTRAVENOUS	INTRAPLEURAL	INTRAPERITONEAL	INTRAMUSCULAR	SUBCUTANEOUS
3.33	1.7 (4)	4 (5)	90 (6)	162 (5)	167 (5)

All the animals died.
The mice weighed between 25 and 30 Gm.

TABLE VII

TIME ELAPSING (IN SECONDS) AFTER INJECTION OF CORAMINE INTO GUINEA-PIGS BEFORE ONSET OF CONVULSIONS. LETHAL EFFECT OF VARYING DOSES ACCORDING TO ROUTE OF ADMINISTRATION

DOSE IN MG. PER KG.	ROUTE OF ADMINISTRATION				
	INTRAVENOUS	INTRAPLEURAL	INTRAPERITONEAL	INTRAMUSCULAR	SUBCUTANEOUS
1.25	7 D	20(2) D*	143(2)	No	No
2.5	4 D	21(3) D	180 D	900 D	1260 D
3.75	4 D	21 D	60 D	360 D	330 D

*One of the two animals died.

TABLE VIII

TIME ELAPSING (IN SECONDS) AFTER INJECTION OF CORAMINE INTO MICE BEFORE ONSET OF CONVULSIONS

DOSE IN MG. PER ANIMAL	ROUTE OF ADMINISTRATION				
	INTRAVENOUS	INTRAPLEURAL	INTRAPERITONEAL	INTRAMUSCULAR	SUBCUTANEOUS
25	2 (3)	4 (3)	100 (3)	350 (2)	310 (2)

All the animals died.
The mice weighed between 25 and 30 Gm.

TABLE IX

TIME ELAPSING (IN SECONDS) AFTER INJECTION OF NEMBUTAL INTO MICE BEFORE ONSET OF SLEEP

ROUTE OF ADMINISTRATION				
INTRAVENOUS	INTRAPLEURAL	INTRAPERITONEAL	INTRAMUSCULAR	SUBCUTANEOUS
1 (4)	40 (5)	120 (5)	182 (5)	280 (4)

The mice weighed between 25 and 30 Gm.
Each animal was given 2.5 mg.

rapid after intrapleural injections, somewhat less rapid after intraperitoneal injections, still slower after intramuscular injections, and slowest after subcutaneous injections.

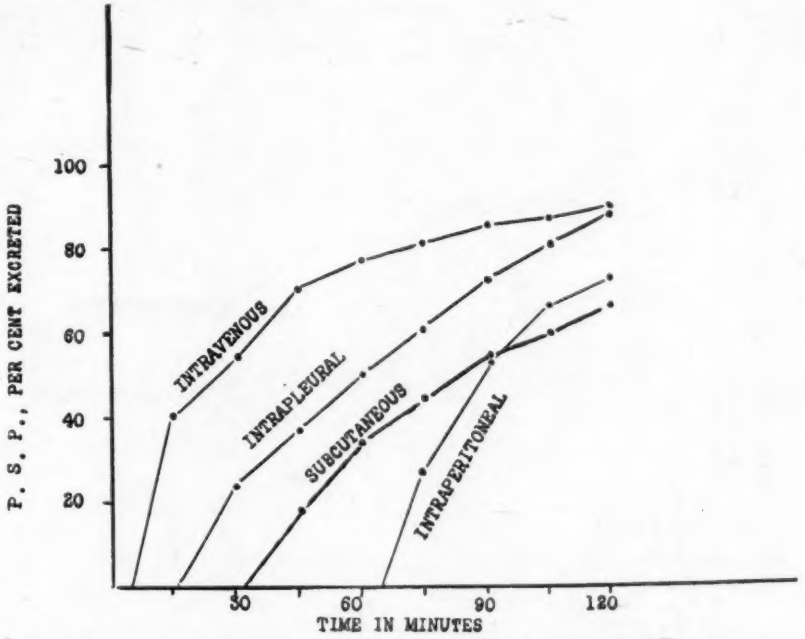

Fig. 1.—Excretion curves of phenolsulfonphthalein administered by various routes. The appearance time is indicated by the point at which the curve begins.

Sulfanilamide.—Three normal healthy mongrel dogs previously starved for twenty-four hours were anesthetized with ether and given 100 mg. of finely powdered sulfanilamide per kilogram of body weight. In one the powder was put into the left thoracic cavity through an intercostal incision. The lung was re-expanded and the wound closed. In a second dog the sulfanilamide was similarly introduced into the peritoneal cavity through a midline incision, and in the third it was placed in a large subcutaneous pocket made in the left upper quadrant of the abdomen. Specimens of blood were taken frequently for assay by the technique of Bratton and Marshall.[2] As recorded in Fig. 2, absorption was most rapid from the pleura and least rapid from the subcutaneous tissues. For example, in thirty-five minutes the concentration in the blood was 12.5 mg. per cent in the dog given the drug intrapleurally, 3.48 mg. per cent in the dog given the drug intraperitoneally, and 0.598 mg. per cent in the one given the drug subcutaneously.

Haag, Spealman, and McCue[3] recently conducted some studies on the absorption of sulfanilamide in dogs and concluded that absorption is as good from the pleura and peritoneum as from the gastrointestinal tract. Although they did not do blood assays in the early period after administration of the drug as frequently as I did, their results also suggest that

absorption is actually more rapid from the pleura than from the peritoneum or the gastrointestinal tract.

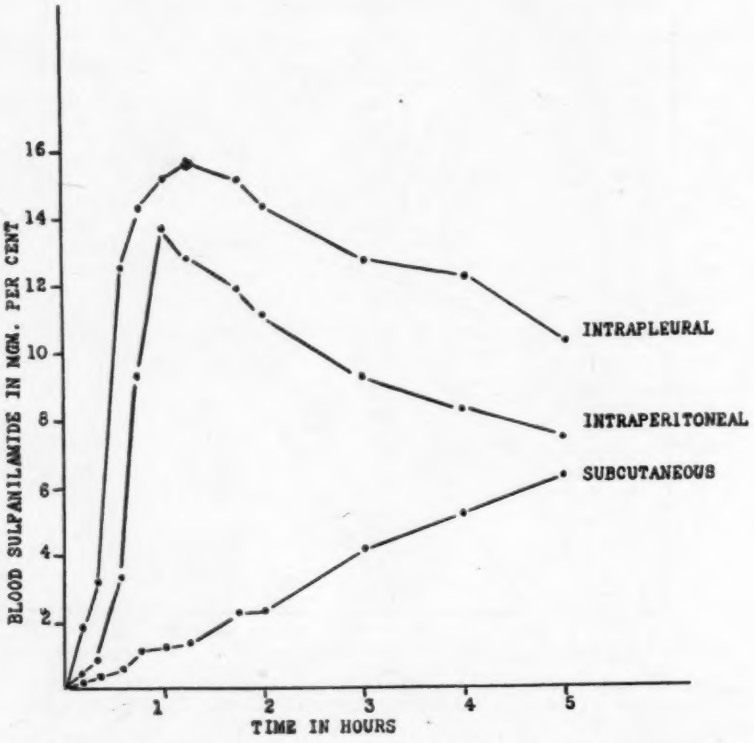

Fig. 2.—Absorption curves of sulfanilamide given by different routes.

DISCUSSION

The present study is by no means complete or exhaustive, and many questions arise. I have made no attempt to determine the relative roles of lymphatic and capillary absorption, the influence of depth and rapidity of respirations on the rate of absorption from pleura and peritoneum, and the question of the size of the molecule in determining whether peritoneal absorption is rapid or very slow. One wonders whether the slow absorption from the peritoneal cavity may result in part from absorption of some of the injected material into the portal circulation with retention or actual chemical change of the drug in the liver. I do not know what influence a local inflammatory process in the pleura or the peritoneum has upon the rate of absorption. These and other matters deserve further study. The experiments reported do demonstrate that with all the drugs studied there is apparently very great difference in the rapidity of absorption from various body tissues and serous cavities. They demonstrate further that with all the drugs studied and in all the species of animals employed, absorption from the pleura is very much more rapid than from any other tissue investigated.

With some drugs peritoneal absorption is relatively rapid, with others very slow.

These studies permit one to emphasize the importance of investigating adequately the rate of absorption of any drug from all the body tissues into which it might be purposefully or inadvertently introduced. This investigation is necessary in order to obtain the best therapeutic effect and to avoid toxic reactions. Such studies should lead to better clinical results and to fewer alarming and even fatal reactions.

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RECONSTRUCTION FOR TRAUMATIC DENUDATION OF THE PENIS AND SCROTUM

Report of a Case

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DEFECTS exhibiting total loss of the skin covering the penis or scrotum, or both, invariably cause grave concern for the patient and frequently present difficult problems of reconstruction for the surgeon who assumes their correction. Fortunately, these accidents occur rarely, there being thirty-four reports in the literature,[1-34] only thirteen[1, 9, 12, 13, 14, 15, 21, 23, 24, 26, 30, 31, 33] of which are on avulsion of the skin of both the penis and scrotum. The earliest report of complete destruction of the skin of the penis and scrotum is that of Gibbs,[34] in 1855. Sixteen years later, Conrad[33] reported a case of reproduction of the scrotum. The remaining reports have all been recorded in the present century. It is interesting to note that with the advent of the more extensive use of machinery, reports of this accident have appeared with increasing frequency.

Defects resulting in complete ablation of skin covering of the penis may be considered either traumatic or infectious. The traumatic group includes those cases in which the loss has resulted from direct trauma, as in accidents[4, 7, 8, 11, 14, 15, 22, 24, 25, 27, 28, 33] or burns, or surgical trauma, as in circumcision.[2] The infectious group consists of those cases arising from any infectious process, as lymphogranuloma inguinale[2] or urethritis.[32]

The essential problem in the correction of surface defects of the penis is that of restoration of skin coverage. Surgical reconstruction is modified by the specific type of defect and associated complications. In those cases in which complete loss of skin has resulted from trauma (accidental or surgical), repair can be satisfactorily accomplished following an adequate débridement and the application of a half-thickness skin graft. Restoration of coverage in the infectious case obviously requires more detailed consideration and, naturally, infection must be eliminated first or sufficiently reduced before a graft can be judiciously applied. The defect must be in a state which will not only permit the formation of healthy granulation but which will also support the graft and maintain its viability.

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In either case the procedure is the same as for repair of any other similar defect where the reconstruction is effected by a half-thickness skin graft. However, it can readily be seen that reconstruction is more difficult here because of the necessity of grafting over a rounded surface. Because of the potential variation in size encountered as a result of normal physiologic response, the problem of maintaining proper apposition of the graft is again exaggerated. These difficulties, however, can be overcome largely by procuring a half-thickness skin graft large enough to encase completely the entire circumference of the penis. External urethrotomy[22] and suprapubic cystotomy have been suggested as preliminary procedures, but are unnecessary. Cases of complete traumatic avulsion of the skin of the penis and scrotum which were seen by a surgeon within the first few hours following the accident have been reported. In some of these repair was attempted after treating the patient for two or three days.[15, 27] Grafting was not attempted in one case[14] until ten days after the injury. Because of the excessive contamination to which a wound is subjected in this region, it is imperative to attempt to obtain primary healing in all cases. This is possible only if the surgeon sees the patient within the contaminated period and the patient's general condition permits an adequate débridement. A graft should then be applied immediately. If these precautions are taken, a full take of the graft with satisfactory healing by primary union may be expected. Following débridement, the graft is wrapped about the penis and attached either by interrupted or continuous sutures along the margins of the defect at or near the corona and toward the base of the penis. In order that firm fixation may be obtained, the graft then is attached to the shaft of the penis down the line where its two borders overlap. Either a grease or moist saline solution dressing of fine mesh gauze is applied directly to the surface of the graft and adjacent to this a few thicknesses of sterile dry gauze are placed. Over these a sea sponge or sterile waste pressure dressing is applied in order that adequate and uniformly distributed pressure may be maintained along the entire shaft of the penis. The pressure dressing is fixed with adhesive, which not only encircles the dressing over the shaft of the penis but extends from the shaft up to the pubis and over the abdomen, thus supporting the dressing.

Other methods of repairing surface defects of the penis have been suggested and used, such as scrotal flap,[19] single pedicle skin transplant, and tube pedicle transplant. Whereas the writer has had no occasion to employ the latter two methods, on one occasion repair of a surface defect of the penis was attempted by means of a pedicle formed by dissecting a cuff of scrotal skin and attaching the penis beneath this so that the under surface of the scrotal skin cuff was in intimate apposi-

tion with the defect over the shaft of the penis. Restoration was successful in this instance and the patient suffered no pain following reconstruction. A report of this case follows.

CASE REPORT

T. B., a 56-year-old negro farmer, was admitted to Charity Hospital, Sept. 3, 1934, on the genitourinary service, complaining of swelling of the penis. Three days before, the penis became swollen rather suddenly. He had had a discharge from the urethra for several weeks. He could urinate only in small streams and complained of a burning sensation following urination.

Fig. 1.

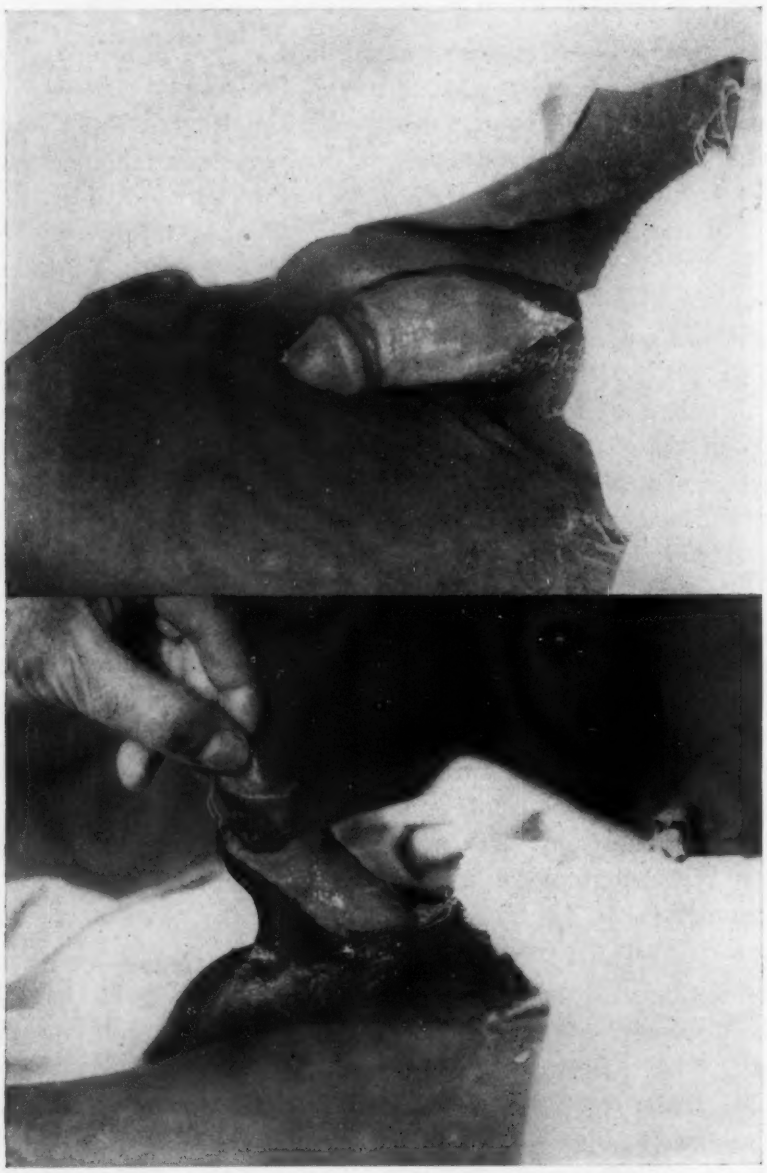

Fig. 2.

Fig. 1.—Photograph showing defect of the penis following infection.
Fig. 2.—Lateral view showing defect of penis following infection.

Examination revealed a greatly enlarged and edematous penis. The edematous prepuce was hanging over the meatus. A urethral discharge was present. The testicles were slightly swollen and tender.

A diagnosis of gangrene involving the whole dorsal portion of the skin of the penis and subcutaneous tissues was made, September 5. On the same day, a suprapubic cystotomy was done by the urologist.

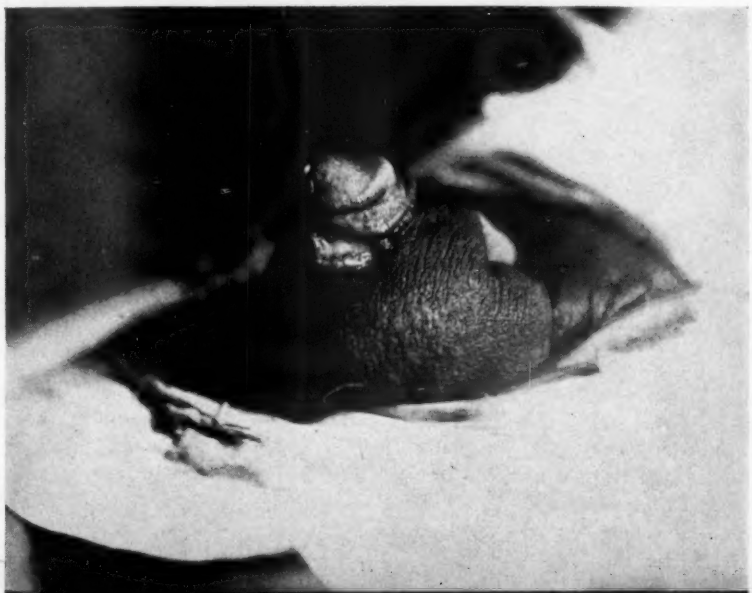

Fig. 3.

Fig. 4.

Fig. 3.—Photograph showing first stage repair where penis is buried beneath scrotal cuff transplant.

Fig. 4.—Photograph showing healing following application of scrotal cuff transplant. A clear comparison of the results obtained from skin graft and scrotal cuff transplant is seen when comparing Fig. 4 and 7.

On October 10, the patient was seen in consultation. An examination at this time revealed a defect of the penis which extended from the corona to the symphysis pubis and involved the entire dorsum to extend downward on either side practically to the frenum, thus leaving only a small portion of skin extending from the frenum back to the scrotum on the under surface. There was evidence of chronic infection and lymphedema in the skin at the base of the penis, which extended down to be reflected in the skin of the scrotum. The defect under the penis was relatively free of infection and under the entire surface there was a firm, smooth, velvety granulation (Figs. 1 and 2).

It was decided to attempt repair with cuff pedicle skin transplant. A pattern of the denuded portion of the penis was made and an area this size outlined over the anterior surface of the scrotum. Transverse lines were made at the point representing the length of the pattern and the skin was incised along these lines down to the subcutaneous tissues. The skin between the two incisions was then separated from the subcutaneous tissues so as to form a tunnel, through which the penis was drawn. With the penis in this position, the undersurface of the skin of the tunnel was in approximation with the denuded area on the dorsum of the penis. Skin borders at the base of the penis and at the corona were pared and to these pared borders end-on mattress sutures of chromic 0000 silkworm gut were applied. A xeroform dressing was then placed over the wound (Fig. 3).

The cuff took nicely and on October 24, the second stage of the operation was done. Under spinal anesthesia, the right pedicle of the cuff was cut and the edges of the penile skin were freed and the cuff sutured to them. On October 31, under local anesthesia, the left pedicle was cut away and the graft sutured to the skin of the penis after it had been freed. The patient was discharged Jan. 2, 1935, completely healed (Fig. 4).

The end result from a scrotal skin flap is not as satisfactory as from the half-thickness skin graft because of the increased thickness and the loss of pliability resulting from the former method.

Because there was complete loss of all scrotal skin and because the defect was further complicated by an extensive laceration downward into the perineum extending almost to the anus, reconstruction of the defect of the penis by a half-thickness skin graft was employed in the following case.

REPORT OF CASE

J. H., a white farmer, aged 38 years, was admitted to Charity Hospital, Oct. 6, 1938. Eight hours before, while working on a rice binder, he stepped too close to the drive shaft connecting the binder to a tractor. Not only the clothing but also the external genitalia were caught by a coupling on the shaft, which was revolving at high speed, resulting in complete denudation of the penis and scrotum (Figs. 5 and 6).

Examination on admission revealed a patient lying flat on his back, fully conscious, with no suggestion of shock. The penis and testicles were wrapped in a dressing of old linen, which was saturated with blood. Close inspection revealed a penis completely denuded from the base to the corona, where there was a small cuff of mucosa. All scrotal skin was lost. The testicles were freely movable and could be widely separated without difficulty. There was no apparent injury to the cord on either side. Beneath the testicles and extending posteriorly almost to the anus, was a laceration which permitted sufficient separation of skin margins to give a large, exposed perineal wound. There was no apparent involvement of the anus nor perforation of the fascia.

It was obvious that skin graft would not be desirable for complete repair of such an extensive defect (entire loss of the skin of the scrotum and penis) because of the inability of adequately applying pressure to hold the skin graft in intimate contact with the total surface of the defect. To attempt to cover the testicles completely by means of single pedicle skin transplant or other types of tissue transplant would have been equally as injudicious. Obviously, satisfactory repair could result only from a procedure which would permit the immediate grafting of the penis and the burial of the testicles under a protective skin covering. Unless this could be accomplished, certain infection with its subsequent complications would ensue. Therefore, the skin was dissected along the medial aspect of each thigh so

Fig. 5.

Fig. 6.

Fig. 5.—Photograph showing complete loss of skin over penis and testicles.

Fig. 6.—Photograph showing complete loss of skin over penis and testicles. Note the complete freedom of the testicles and the extensive lacerated wound shown below the right testicle where it extends deep into the perineum.

as to form a small pocket between the skin and the underlying fat and fascia of the thigh. This was enlarged sufficiently to permit the reception of a single testicle, each being placed in a corresponding pocket on its respective thigh. There was no undue tension nor distortion of the testicles. On the contrary, the pocket seemed to support the testicles so that there was less tension on the cord than is usually seen. The skin border above the pocket was then approximated with the opposite skin border of the defect and held together by means of interrupted sutures of black silk and 0000 silkworm gut. Thus, each testicle was completely encased beneath the skin, medial to the thigh on its respective side, and was covered with skin and a

Fig. 7.

Fig. 8.

Fig. 7.—Photograph showing healing following the application of skin graft to the penis. Photograph taken approximately three months following operation.

Fig. 8.—Photograph showing complete healing of penis following application of skin graft. This photograph shows the testicles where they are imbedded beneath the skin of the thighs as shown by the outline over the skin of the thighs.

slight padding of fat as a protection. A half-thickness skin graft was removed from the inner surface of the right arm and applied around the entire shaft of the penis. It was held fixed by means of interrupted sutures which completely encircled the skin near the corona and another row of interrupted sutures attaching the skin graft to the abdominal perineal skin at the base of the penis. Sutures were also taken down the line where the longitudinal border of the skin graft overlapped. This held the skin firmly in place around the shaft of the penis. A xeroform gauze dressing was applied over the entire surface of the skin graft. Over this a dry gauze, sea-sponge, pressure dressing was applied. A week later the dressing was removed, revealing a satisfactory graft. There was no evidence of infection around the perineal wound nor in the pockets which had been formed for the reception of the two testicles. The dressing was changed daily thereafter, and on the fourteenth day the patient was permitted to sit up in a chair (Figs. 7 and 8).

On the theory that sterility ensues in a testicle which maintains a temperature equal to body heat and not one degree less, reconstruction of a scrotum was next considered. This could be done by forming a single pedicle skin transplant from the skin of the thigh immediately covering the buried testicle and raising the skin and its underlying fat with the testicle attached so as to bring it into approximation with a similar flap on the opposite side.[14] The two flaps covering their respective testicles would meet and approximate in a cuplike fashion so that they could be sutured around their circumference, thus completely encircling the two testicles and giving absolute skin covering with an underlying fat pad for protection. This, of course, would have necessitated three operative stages. The resulting scrotal sac would have been considerably larger than normal and would have been devoid of any contractile property. However, the patient was not even remotely interested in this operation. When confronted with the fact that he would most likely be sterile if some attempt to remove the testicles from their present site was not carried out, he stated that sterility was desirable because of the large size of his family. When last seen and examined, April 1, 1940, the patient was found to be completely healed with satisfactory results. He has experienced no difficulty in any respect and there has been no disturbance whatever with normal physiologic functions.

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THE ELEVATION OF THE NASAL BRIDGE LINE

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INTRODUCTION

FOR centuries the stigma of a misshapen nose has caused the bearer to seek relief of his deformity either through surgery or through the camouflage of a corrective cosmetic make-up. In the early middle ages, artificial noses were often worn by normal people as a stylish fetish. Therefore, little comment was caused when those with nasal defects wore these same artificial noses to cover their deformities. In this age the so-called "fathers of plastic surgery" rose to fame, not by their ability to reconstruct an ear or to graft an unhealed area, but primarly on the self-heralded excellence of their nasal reconstructions. Their claims of success became so fantastic that they excited the ridicule and disbelief of both the church and the public, so that by the late eighteenth century the whole field of reconstructive surgery had fallen into great disrepute. Such a stage of indignation over charlatan claims and practices was reached that the Paris Faculty in 1778 passed a resolution forbidding any further corrective surgery of the face.

Since that time the scope of plastic surgery has widened to include the relief of many conditions other than nasal deformities. The principles governing this field of surgery have been founded gradually and, contrary to popular belief, were well established long before World War I. Several procedures were tested during this war which resulted in the popular adoption of many of the sound procedures and the discontinuation of most of the unsound ones. These principles have been embellished further during peace time so that now they have attained significant stature among the surgical fundamentals for World War II.

CAUSES OF NASAL DEPRESSION

Congenital.—The external portion of the nose is formed by a central field of tissue that curves down over the head of the embryo and advances caudally on the ventral surface until it meets the paired maxillary protuberances from the first branchial arch to form the upper lip. This central cranial field, more commonly called the nasal field, is made up of four anlage, a central pair bounded on each side by another which

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together make the lateral pair. The central pair form a portion of the dorsum of the nose, a portion of the nasal septum, the columella and filtrum of the lip. The lateral pair develop into the sides of the nose and the alae.

If there is an absence of the entire nasal field, the patient is born with no nose at all. If there is an arrest in its descent, the nose is represented by two nostrils up above or between the eyes. If one lateral nasal process fails to descend, the patient will be born with only one nostril.

If the descent of these processes progresses in the normal manner but the amount of tissue contributed by each is deficient, the result is a perfectly formed nose except that it is too small. A patient with this anomaly characteristically has a very flat nasal bridge, epicanthal folds, and a turned up nubbin of soft tissue perforated with normal nostrils representing the tip. It is this latter type of deformity that is ordinarily neglected when the patient is a child because of the erroneous idea that it will correct itself. This results in a young adult with a nose many sizes too small for the rest of the face. It is very difficult to correct the deformity in adult life because the soft tissue, deficient to begin with and never having been stretched by proper bone growth beneath it, is incapable of being distended to the proper size in one operative maneuver. It is my opinion that an osteochondral graft from the crest of the ilium should be inserted when the patient is 4 or 5 years of age in an effort to stretch this skin early in life. If the graft takes properly it should presumably grow with the patient, as is shown in Case 1. If the result is satisfactory when the patient reaches 15 or 16 years of age, further operative procedures will not be necessary. If it is not satisfactory, this first graft can either be added to, or removed and replaced with a larger graft at this time. The procedure of grafting early should produce gradual stretching of the skin covering so the insertion of a graft of adequate size can more easily be accomplished when the face has reached adult proportions.

Neoplastic.—Tumors involving the nose, like those elsewhere on the body, may be either benign or malignant. Benign tumors, such as hemangiomas or lymphangiomas, are usually eradicated by radiation or excision without the destruction of the bony framework. Malignant tumors may involve bone and thus require excision of the entire nose including the bony and cartilaginous framework to remove the tumor successfully. In the latter case an entire nose must be rebuilt, a procedure which does not come within the scope of this paper. Benign tumors of the septum, such as a dermoid, may be so extensive that removal of all or part of the supporting structures may be necessary for extirpation of the growth. The late result may be either a depressed nasal bridge or tip which will require elevation with a graft for a good cosmetic effect.

Infections.—Infections frequently follow trauma to the nose in which there has been a compound fracture of either bone or cartilage, or in

which a hematoma, particularly of the septum, is allowed to remain undrained so that it subsequently becomes infected. An osteomyelitis of the nasal bones is the frequent sequelae with either absorption or extru-

Fig. 1. Fig. 2.

Figs. 1 and 2 (Case 1).—This patient exhibits a congenitally small nose with the deficiency particularly marked at the nasal bridge. She was observed over a period of two years during which time there was no improvement. At the age of six years a hinged osteochondral graft was inserted.

Fig. 3. Fig. 4.

Figs. 3 and 4 (Case 1).—Photographs taken two years after insertion of the hinged osteochondral graft. There has been definite growth of the graft in proportion to the rest of the body growth.

sion of the bone as sequestra. Infection of the hair follicles within the nasal vestibule may extend to the septum to cause destruction of the cartilaginous portion of the septum and occasionally the bony framework. For centuries, lupus and syphilis have been classic in producing nasal deformities which were so distinctive that they were easily recognized even by laymen.

Fig. 5. Fig. 6.

Figs. 5 and 6 (Case 2).—This patient received a crushing injury to his nose which resulted in a compound fracture of the nasal bones and septum. Following this he had an infection of the septum (cartilaginous) which subsided after drainage. These photographs were taken one year after the injury.

The successful treatment of nasal destruction following infection depends upon the complete control of whatever infectious organisms caused the original deformity. Syphilis and lupus must, therefore, be well bridled before reparative measures are begun. To allay the danger of reactivating an old infection it is always a wise precaution to wait at least six months to a year after an acute infectious process has subsided before proceeding with operative reconstruction. It is also necessary to evaluate exactly what tissues have been lost before the repair is begun. If a significant portion of either the nasal skin or the nasal mucosa has been destroyed in addition to the bony or cartilaginous support, no amount of stretching of the remainder by means of a graft will produce a satisfactory result. In the latter case a lined pedicle flap from the forehead, arm, or acromiopectoral region is brought up to replace the loss of soft tissue and at a later date the supporting structure is inserted.

Trauma.—Fractures of the supporting framework of the nose are frequent and are accompanied by varying degrees of displacement of these structures. Unless the comminution of the fragments is excessive, all of the parts that are displaced usually can be restored to their original positions soon after the injury without the necessity of using a graft for additional support. However, if the trauma has been frequent and repeated at intervals over a prolonged period of time, simple refracturing and restoration of the bone and cartilage to their normal positions often is not sufficient to produce a good cosmetic result. These old fractures are characterized by a flattening of the entire nasal bridge in addition to a crushed and badly deviated septum which is

Fig. 7. Fig. 8.

Figs. 7 and 8 (Case 2).—Photographs taken eight months after insertion of a single wedge-shaped bone graft from the iliac crest without the use of the septal hinged portion.

not able to be permanently raised or straightened. In the latter case it is preferable to perform a submucous resection of the distorted septum and, possibly during the same operative procedure, to refracture and set the nasal bones into as nearly normal a position as possible. After a six-month interval further elevation of the bridge line can be accomplished by the insertion of a graft. In some patients whose features are naturally large, the flattening of the nasal bones may be so marked that it is frequently more satisfactory to build a broader nose by inserting a wide graft without the preliminary refracturing (Case 4).

CHOICE OF MATERIALS

The methods of elevation of the depressed nasal bridge line have run the plastic gamut perhaps more than any other reconstructive procedure. After the passing of the era of artificial noses, attempts were made to fill up a nasal depression by the insertion of various nonviable

Fig. 9.

Fig. 10.

Figs. 9 and 10 (Case 3).—Photographs taken fourteen years after an operation for a deviated septum. At that time either too much of the septum was removed or the patient developed a hematoma that became infected and resulted in this depression.

Fig. 11.

Fig. 12.

Figs. 11 and 12 (Case 3).—Photographs taken two years after insertion of a hinged bone graft taken from the crest of the ilium.

substances under the nasal skin. Of these, the most widely used was a wax of low melting point which was injected in the subcutaneous tissue over the nose, moulded in the proper shape with the fingers, and set by the application of cold packs. The effect was only temporary, the usual termination being either a slough of the nasal skin or a paraffinoma. It is unfortunate that this method is still used by less reputable men and some licensed cosmeticians. Other inert substances such as silver, celluloid, and ivory, when cut to the proper shape, have been and are still used. These artificial supports have usually caused the surrounding tissues to react in such a fashion that extrusion of the foreign body was inevitable.

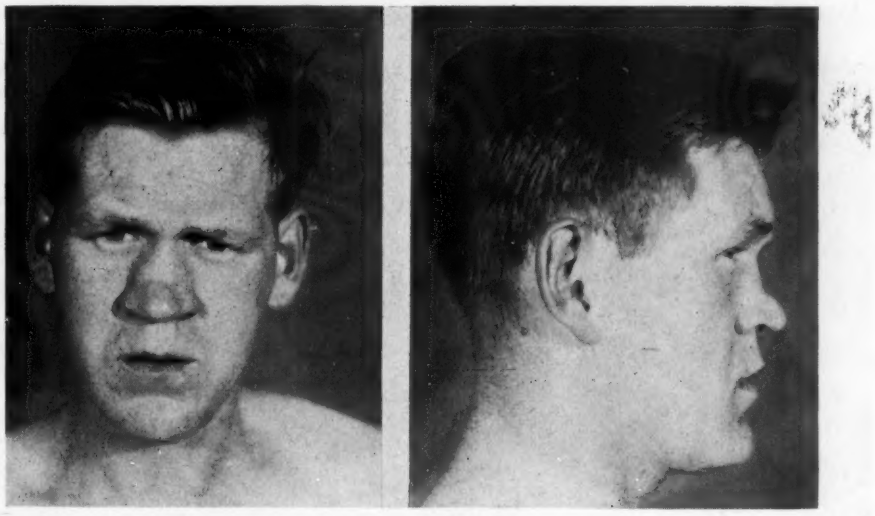

Fig. 13. Fig. 14.

Figs. 13 and 14 (Case 4).—Photographs of a patient who had had his nose fractured on five different occasions in eleven years. The last injury occurred two years before these pictures were taken.

Autogenous grafts of fat or fascia have been employed with varying degrees of success. A sufficient residue of either tissue is difficult to maintain, as uncertain proportions of both substances tend to melt away gradually when transplanted out of their usual environment. Through trial and error, the materials insuring the best and most permanent results have been found to be either bone or cartilage. The autogenous grafts of both of these substances are used by the majority of plastic surgeons, but recently preserved homogenous grafts of cartilage have been found to be equally successful.*

The author has found that the use of autogenous cartilage grafts has one important disadvantage to offset the obvious advantage of working with a material that may be easily shaped to the desired contour. The only donor site of autogenous cartilage of the desired

*C. L. Straith, and W. B. Slaughter, M.D.: Grafts of Preserved Cartilage in Restoration of Facial Contour, J. A. M. A. 116: 2008-2013, 1941.

quantity necessary for raising the nasal bridge is situated at the juncture of the anterior ribcage with the sternum. All of these cartilages are naturally curved. In order to fashion a straight segment from this tissue it is necessary to whittle it down at both ends and leave the middle relatively undisturbed. Microscopic examination of the finished cartilaginous strut, even though straight externally, will show that the predominance of cells still run in a curved direction. When this support is placed under the nasal skin it will appear at first to be straight but as time goes on, an appreciable number of grafts will begin to twist and re-establish the original curvature that existed in the donor sites. It has been stated that preserved homogenous grafts have less of this distressing tendency toward the development of curvatures subsequent to operation, than either homogenous or autogenous fresh cartilage.

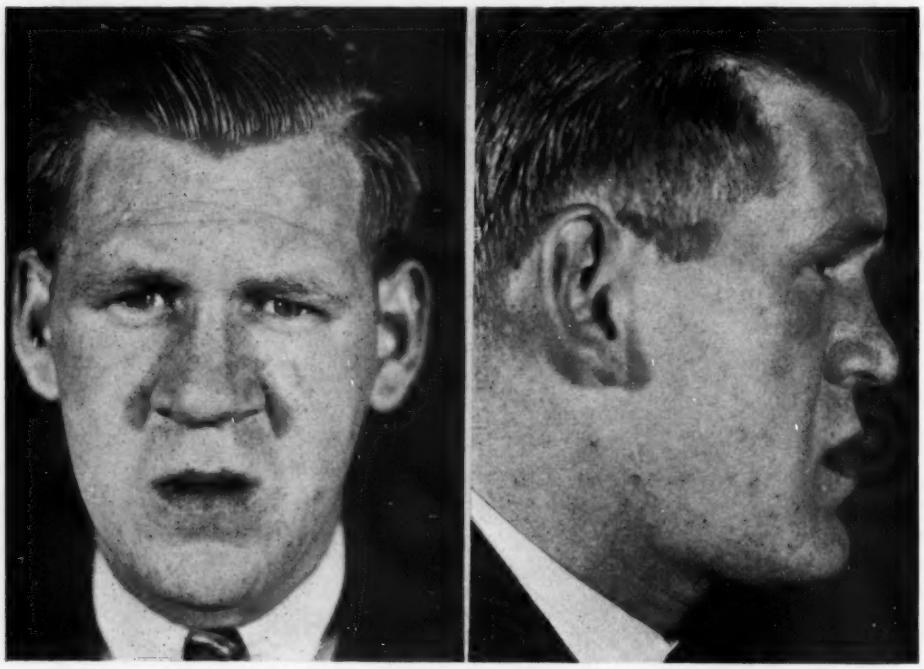

Fig. 15. Fig. 16.

Figs. 15 and 16 (Case 4).—Photographs taken one month after insertion of a hinged bone graft from the crest of the ilium. The entire width of the iliac crest was used to fashion a wide enough graft so that the new nose would match the other rugged features in this face.

When bone is properly used for nasal support this disadvantage of late curvature is eliminated. Some authors have reported gradual absorption of calcium from the bony framework in this site and the replacement of it with fibrous tissue. However, this fibrous tissue replacement has usually been in quantities sufficient to retain the original cosmetic effect so that the loss of calcium is of little importance. This absorption has not taken place in those cases in which the graft was inserted in such a fashion that bony union between the graft and the nasal bridge was the effect wished for and obtained.

The desired amount of bone necessary for this reconstructive work is more easily available than is a similar amount of cartilage and may be procured from either the iliac crest, tibial crest, or ribs. The only distinct drawback encountered with the usage of bony struts is the difficulty in handling and carving it to the desired shape. This latter difficulty may be overcome by use of the proper instruments and the development of an operative technique which is suitable. It has been my experience that bone from the iliac crest has served admirably to relieve nasal deformity without any sequelae that were distressing to either the patient or surgeon.

TECHNIQUE OF OBTAINING AND USING ILIAC BONE

On the evening before operation the hairs inside both nostrils are clipped and the nasal vestibule cleaned with swabs of normal saline solution. The face is then thoroughly washed with mild soap and water and swabbed with a weak astringent solution. In women, the use of face creams usually applied upon retiring is forbidden. The pubic area, lower abdomen, and upper thighs are shaved and cleansed in the same fashion as the face. These fields are not wrapped in sterile towels, as I believe that this practice produces an excess in perspiration, thereby raising the local bacterial count. On the following morning all fields are again cleansed in the same fashion as they were the night before, except that for men a complete close shave of the face is done two hours before operation.

Although the entire operation is possible under a local anesthetic to the nasal area and a spinal anesthetic for the hip area, I advise a general anesthetic, preferably rectal avertin supplemented with intratracheal gas-oxygen ether. A general anesthetic eliminates any psychic distress due to the noise and pressure caused by the chiselling of the bone from the iliac crest.

At the time of the operation the fields are again prepared with soap, water, alcohol, and ether. The inside of the nose is carefully swabbed with some mild nonirritating antiseptic such as zephiran or aqueous merthiolate. The distance is now measured off from the glabella to the apex of the nasal tip, a point formed by the anterior reflexion of the alar cartilages on the septal cartilages. These two points are marked with Bonney's blue paint and the midline of the nose between these points is dotted on the skin with a sharp fine needle dipped in the same dye. If only a single straight wedge-shaped graft is to be used without a columellar strut this glabella-to-tip measurement will then represent the length of the desired bony segment. If a columellar strut is needed to insure support of the tip, then this measurement represents only two-thirds of the length of the required amount of bone.

A solution of eight drops of $\frac{1}{1,000}$ adrenalin to the ounce of either saline solution or novocain is injected next into the columella, the

septum, and beneath the periosteum of the nasal bones. This will facilitate both the dissection and the control of hemorrhage. A vertical incision is made through the skin of the columella, dividing it into two longitudinal halves. To prevent perforation of the septal mucosa a forefinger is inserted in one nostril and the thumb in the other; the lower septum is held between the two while the skin incision is deepened by sharp dissection until the knife reaches the nasal bones. The operator then changes the glove on the hand that held the septum and packs each nostril with a swab soaked in a mild antiseptic solution. The knife is again inserted and the periosteum of the nasal bones is cut transversely at its anterior attachment with the bones. A periosteal elevator is then inserted through the columellar incision, and guided through the transverse incision in the periosteum, where it is pushed upward carefully in order to raise the periosteum off the bones and create an oblong pocket all the way up to the glabella. After this pocket has been formed, the outer table of the nasal bones are scraped with a file to give a better foundation for the graft. The entire cavity thus formed is now packed with dry gauze to control hemorrhage. Care must be taken in placing this pack to prevent distention of the skin to the point of shutting off the circulation to the relatively thin layer over the bridge line. A pack of iced sterile saline solution is placed over the outside of the nose to help control hemorrhage and to prevent excessive edema. It must be emphasized that this dissection should be done most gently and carefully, with the tips of the fingers of one hand always placed on the external surface of the nose over the point where the instrument is being used. This is the only safe way to prevent tearing through or "buttonholing" the skin over the bridge line.

An incision is now made over the iliac crest so that the inferior end of the incision terminates at the anterosuperior spine of the ilium. The incision is carried down to, but not through, the periosteum. The attachment of the muscles is freed from the lateral border of the crest for the entire length of the incision and for a depth of about one inch. Bleeding is usually controlled by retraction of the cut muscle ends under pressure. The desired length of bone is now measured off on the iliac crest. Usually the ilium is of sufficient thickness so that only a portion of the crest and outer table is needed. Occasionally a wide nasal bridge is required so that a segment comprised of both the inner and outer tables is removed in one piece (Case 4).

The procedure of removing this segment must follow a strictly "no-hand-touch" technique. Separate instruments and instrument tables are used to avoid any possible contamination of the raw bone with gloved hands that have come in contact with the external skin. The bony fragment is chiseled away from the crest and is then dropped into sterile saline solution while the donor site is closed. The closure is in three layers with either interrupted fine catgut sutures or fine

silk sutures to the muscle and fascia layers, subcutaneous layer, and subcuticular layer. A continuous suture of fine dermic is used for the skin. A small rubber wick drain is inserted down to the bone to remain in place for twenty-four hours. A pressure dressing of gauze, elastoplast, and A-S-E bandage is then applied.

The bone removed from the ilium is now sculptured to the desired size and shape. This part of the procedure is the most difficult and exasperating of the entire operation. Unless the instruments used for holding the graft are adequate, the graft is likely to skid off the table. If the instruments used to mold it are dull, it may be broken or crushed out of its planned shape.

If a single bridge strut is to be used, the shape of the graft on anteroposterior view is roughly rectangular, the end of the rectangle which will lie at the nasal tip being slightly narrower than that which will lie at the glabellar end. On the lateral or profile view, this graft is thin in the glabellar region, reaches the limit of its thickness at the point where the graft will leave the support of the nasal bones on which it is to rest, and again gradually thins toward the tip. In this view the part that will form the bridge line is straight, so that one can imagine the graft to be shaped like a solid triangle, the base being the bridge line, one side the portion that will rest on the nasal bones, and the other side the part that extends from the nasal bones to the tip of the nose. The apex or peak of the triangle rests at the point where the graft leaves the support at the anterior end of the nasal bones. The under surface of the side that rests on the nasal bones is grooved slightly down its center to fit tightly to the slight transverse curvature of the nasal bones.

If a columellar strut is needed in addition to the bridge support, a segment of bone one-third longer than that required for a single wedge graft is removed. The bridge portion is formed in the same fashion as that described above but the periosteum of the graft is left intact at the tip end to serve as a hinge for the narrow columellar portion. The columellar portion folds back on the hinge so that the intact periosteum forms an angle of 270° at the tip. Further support of the tip is effected by the upper end of the body of the columellar strut which rests against the slanting under surface of the tip of the bridge portion for a distance of about 1 cm.

After the graft has been cut to the desired shape, the pack is removed from the nose slowly and carefully, to prevent hemorrhage. Two skin hooks are placed in the skin to hold the incision apart while the graft is inserted. It is absolutely necessary that the graft be placed inside and not above the periosteal pocket made to receive it. If the graft has been correctly shaped by measurement it will fit tightly and securely without any tendency to shift or twist out of position. When a columellar prop is used the free end must fit snugly onto the maxillary mens in order to give stability to the tip. The columellar wound

is now closed by sutures of 4-0 plain catgut to the subcutaneous tissue, and the skin approximated with sutures of 8-0 dermic. Whitehead's varnish is painted on the wound, over which is placed a one-half inch gauze tape that will become adherent to the wound when the varnish becomes dry enough to be tacky.

POSTOPERATIVE CARE

For the first forty-eight hours after operation constant sterile iced saline solution compresses are kept over the nose and eyes. Any accumulation of blood in either the donor or recipient areas must be immediately expressed or aspirated to prevent infection and residual thickening in these sites. The skin sutures are removed from the columella on the third or fourth postoperative day and those from the donor wound on the seventh postoperative day. The patient's nostrils are carefully oiled each day and kept free of crusts. When the edema begins to subside on the third or fourth postoperative day the mucosa may be shrunken even further with sprays of ephedrine sulphate. This is done primarily for the patient's comfort and to prevent sneezing or coughing. Both of the latter acts, if violently done, may start up a secondary hemorrhage in the periosteal pocket. The patient is allowed to walk about on the eighth or ninth postoperative day, at which time he usually complains of muscle soreness in the hip region. This soreness generally disappears after a few days of graduated exercise so that the patient can resume normal work two to three weeks after operation.

The graft becomes firmly adherent by bony union to the nasal bones within six to eight weeks. X-rays taken at this and subsequent times show moderate loss of calcification comparable to that which would occur in any bone of the extremities after a period of six to eight weeks of nonuse.

The result may now be considered permanent, because if primary healing is good, subsequent absorption does not occur. Further evidence of the fact that this graft is viable is proved by one of the cases treated by Gillies* in which the bone graft was fractured by a blow to the nose several months after implantation. Simple setting of this fracture of the graft was done. After six weeks the two fractured ends of the graft had healed in good position with the resumption of the previous excellent cosmetic result.

CONCLUSIONS

Bone grafts taken from the iliac crest and inserted in the nose for the purpose of raising the bridge line or giving support to the nasal tip have produced satisfactory results without extrusion, destruction, distortion, or absorption of the graft at a later time. Specific technical details for obtaining, shaping, and inserting this type of graft have been outlined, together with indications for its use.

*Sir Harold Gillies: Personal communication.

COTTON SUTURE MATERIAL AND EARLY AMBULATION IN GYNECOLOGY AND OBSTETRICS

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IN RECENT years there has been a decided trend in surgical practice toward adoption of nonabsorbable suture materials in most fields of endeavor, and of these, silk has been the most widely used. Even more recently, however, stimulated by the reports of Gage, and Meade and Ochsner, spool cotton has acquired prominence. Each month new reports confirm the original observations justifying the use of cotton and indicating its ever-widening acceptance. In gynecologic surgery the swing toward nonabsorbable materials has been less enthusiastic, largely because of prejudice born of misuse of the materials employed. In July, 1941, a clinical trial of spool cotton was instituted in selected cases on the colored gynecologic service, Tulane Unit, Charity Hospital, New Orleans. Results in these first few cases were so gratifying that the scope was rapidly broadened until, at present, cotton suture material is used exclusively by us in all gynecologic operations. We now have complete data on 160 cases, and it is our purpose to report the results in these cases.

Needless to say, in changing from catgut to cotton sutures, our technique had to be altered completely, and parenthetically, considerably improved. Cotton, like any other nonabsorbable suture, when used according to the principles of Halsted, calls for an exactness of technique, no longer the mass crushing and ligation of tissue, with its inevitable tissue necrosis. At first this change was difficult and it seemed almost necessary to continue our former methods of handling the tough ligamentous and scarred tissue. With increased experience, however, we found it simpler to deal with smaller bits of tissue and employ lighter suture material. When our study commenced, for instance, crochet cotton No. 20 was used to ligate the uterine and ovarian vessels; this was quickly reduced to No. 8. Now we are able to perform total hysterectomy with no suture material heavier than No. 40 (ordinary sewing cotton). In plastic work we have reduced the size of our sutures to No. 80 and No. 120, almost incredibly fine in diameter, but with sufficient tensile strength for the purpose. We have found that the average increased

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operating time is about twenty minutes, which would be expected with the use of interrupted sutures, but we feel that this time is well spent, as borne out by the encouraging results that follow.

Among the 115 abdominal incisions there was no single case of serious wound infection, which is noteworthy considering that practically all of the patients were of the lower class, and that the majority were so-called "dirty cases." There was a gross incidence of slight wound infection, which did not interfere with convalescence, in 8.5 per cent including two cases in which subfascial drains were used at the time of surgery as a precautionary measure. In these two cases there was rupture of fresh tubo-ovarian abscess with peritoneal soiling. In no instance was the peritoneal cavity drained. At first this figure may appear to be high but when it is considered that all of our incisions are placed in the midline, arising at the umbilicus and terminating the pubic hair area, which is too often inadequately prepared, we feel that the figure is exceptionally good. Miller quotes an incidence of roughly 20 per cent following abdominal hysterectomy on the combined colored and white Tulane University Services at Charity Hospital during 1939 and 1940.

We believe that we have subjected cotton sutures to a rigid clinical test on our service because of the routine postoperative handling of our patients. All operated cases have a standing order granting bathroom privileges within the first twenty-four hours after surgery. Those patients who do not walk on the evening of the operation are escorted to the bathroom the following morning, remaining ambulatory thereafter. Of course, there are occasional exceptions to this rule, but these should be classed as infrequent and specified exceptions, such as peritonitis and pneumonia. Among the 115 laparotomy operations there were sixty-nine patients with total hysterectomies, the majority of which had one or both adnexa removed in addition to appendectomy in 52 per cent, plastic in 4 per cent, and umbilical hernioplasty in 3 per cent. Stay or retention sutures were never used. As stated previously, there was no incidence of serious wound infection, no wound disruption, and in the follow-up there has been no incidence of incisional hernia, to date, among the cotton cases. It might be mentioned here, that while this study was in progress, there were three instances of noninfected abdominal wound disruption among nonambulatory white patients and one colored patient in whom chromic catgut was used. All of these were secondarily closed with cotton sutures and healed uneventfully with exception of the one colored patient, who died in bed the next day of pulmonary embolus.

Our success in early ambulation, together with improved wound healing, is not attributed entirely to cotton. All patients have routine serum protein determinations and are administered blood or plasma until this figure is normal. Cevitamic acid is administered routinely in large doses pre- and postoperatively.

Our results in plastic procedure are even more gratifying. Here again, we have been reassured by excellent functional and cosmetic results, lowered morbidity and postoperative comfort. As with the laparotomies, the patients are ambulatory in perfect comfort. It is almost in the realm of fancy to see patients who have had such operations as vaginal hysterectomies and repair of third-degree lacerations, walking and sitting at ease within twenty-four to thirty-six hours after operation. In several instances patients thought that the operation had not yet been performed. This is attributed mainly to lack of tissue reaction resulting from cotton sutures and gentleness in technique. Patients who have had vaginal hysterectomy can be comfortably examined three days after surgery; there is a noticeable lack of discharge, perineal edema, and reaction about the sutures. Again, it must be emphasized that this will follow only after careful surgical technique, especially the inclusion of but small bits of tissue in any one suture, and the use of only fine sutures. Among our plastic procedures, healing was per primam in 100 per cent of the cases, with the exception of one fresh third-degree laceration which evinced a small fecal fistula at the top of the repair. In this instance there was no infection and the poor immediate result is attributed to an omissive error in the repair. The fistula closed spontaneously four weeks following operation. Infection has not occurred in any of our plastic procedures. Table I contains a list of the types of cases in which cotton has been successfully employed.

TABLE I

CASES IN WHICH COTTON SUTURE MATERIAL HAS BEEN SUCCESSFULLY EMPLOYED

Hysterectomies	97	Total abdominal	69	71.1%
		Supracervical	6	6.2%
		Vaginal	22	22.7%
Laparotomies	36	Including suspensions, correction of inflammatory conditions, ectopic pregnancies, associated with various plastic procedures.		
Third-degree lacerations	6	Two fresh, four old, two associated with vaginal hysterectomy.		
LeFort colpocleisis	3			
Secondary repair of wound disruption	4			
Manchester	3	(Two with cervical amputation)		
Anterior and posterior colporrhaphy	2			
Cervical amputation	3	(Sturmdorf, two; Schroeder, one)		
Watkins interposition	1			
Vesicovaginal fistula	1			
Inguinal hernia	1			
Caruncle	1			
Episiotomy	2			

In addition to the above cases, there have been many more episiotomies on our obstetrical service (now done routinely with cotton No. 80 to No. 150 on all cases), the detailed reports of which are not yet available. All of these episiotomies have been uncomplicated during the hospital stay and from the results so far seen in our follow-up clinic we feel that

cotton will survive as a suture of choice in this procedure. In all instances there has been notable freedom from incisional discomfort, as indicated by the fact that all patients are instructed to return to the postpartal clinic for examination and insertion of pessary where indicated as soon as the lochia stops. One of us (C. G. C.) has used cotton sutures with excellent results in episiotomies, plastics, vulvectomy, vesicovaginal fistula, and total abdominal and vaginal hysterectomies, in his private work.

Our results in the larger groups of cases are presented in Table II.

TABLE II

OPERATION	TOTAL HYSTERECTOMY	SUPRACERVICAL HYSTERECTOMY	VAGINAL HYSTERECTOMY	LAPAROTOMIES	ANTERIOR AND POSTERIOR PLASTICS	LeFORT	MANCHESTER	THIRD-DEGREE LACERATIONS
Number of cases	69	6	22	36	2	3	3	6
Mortality (%)	2.81	0	0	0	0	0	0	0
Morbidity (%)	24.6	33.3	22.7	27.7	0	33.3	0	33.3
Average hospital stay (days)	9.6	9	9.5	7	6	8.3	7.3	11.5
Increased hospital stay (%)	11.5	0	15.4	0	0	0	0	0
Positive serology (%)	36.2	20	13.6	22.8	0	0	0	16.6
Catheterization (%)	39.1	0	54.5	27.7	0	0	66.6	50
Cystitis (%)	18.8	16.6	36.3	8.5	0	33.3	0	0
Slight wound infection (%)	10.1	0	0	5.5	0	0	0	0
Stitch abscess (%)	2.8	0	0	0	0	0	0	0
Serious wound infection	0	0	0	0	0	0	0	0
"Cold" (%)	7.2	0	4.5	0	0	0	0	0
Pneumonia (%)	0	0	4.5	0	0	0	0	0
Infected cuff (%)	4.3	0	4.5	0	0	0	0	0
Peritonitis (%)	2.8	0	0	0	0	0	0	0
Pulmonary embolus	0	0	0	0	0	0	0	0
Thrombophlebitis	0	0	0	0	0	0	0	0
Hematoma (%)	1.4	0	4.5	3.7	0	0	0	0
Postoperative bleeding (%)	0	0	4.5	0	0	0	0	0
Fistula	0	0	0	0	0	0	0	0
Follow-up (%)	98.5	33.3	54.5	55	2	100	66.6	83.3

The two deaths in the total hysterectomy series were both due to *Bacillus welchii* peritonitis, proved at autopsy. The source of infection could not be positively traced. One patient was operated upon for large fibroids and carcinoma in situ of the cervix discovered on microscopic examination; this patient died six days after operation. The other patient had a badly infected cervix and extensive pelvic disease; she died forty-eight hours postoperatively. While both patients had an appendectomy, nevertheless, it is felt that the cervix was the source of contamination. Aerobic and anerobic culture studies of our suture material failed to show any growth, excluding this possible source of contamination.

The patients exhibiting positive blood serology for syphilis showed no difference in their convalescence, except that the 7.1 per cent incidence of small granulations at the roof of the vaginal vault following total hysterectomy at six weeks, all occurred in syphilitic cases. Of the vaginal hysterectomies, 9.5 per cent (two cases) showed granulation; 50 per cent of these were syphilitic. This granulation disappeared within four weeks after topical application of silver nitrate. Of course the same type of granulation tissue in the vaginal wall is seen following the use of catgut in a similar operation.

Among the third-degree lacerations, it might be mentioned that one patient weighed 280 pounds and had a vaginal hysterectomy together with Ristine-Nobel repair of a previously repaired third-degree tear. This patient was ambulatory within twenty-four hours and had a 100 per cent functional and cosmetic result.

Criticism may be made regarding the early ambulation of our patients. This practice was stimulated by our previous incidence of thrombophlebitis and pulmonary embolism. Several European authors have described the successful employment of early ambulation, and more recently Ochsner and DeBakey have recommended its use in the prevention of these complications. While our series is yet small, we have confidence that we will be successfully rewarded in our efforts to prevent these two major postoperative complications. In addition, we have observed a much more rapid convalescence, absence of postoperative weakness, quicker return of bowel and bladder function, and lessened morbidity in general. Without the assurance of nonabsorbable sutures we would be reluctant to allow patients out of bed so quickly.

It has been our practice to leave in situ all sutures used in plastic work in order to determine the fate of these sutures. We have observed them in the follow-up clinic over a period of six months; the knots remain visible and there is no reaction at the site of the sutures. Some of these sutures are undoubtedly shed, but the patients are unaware of their presence or passage.

SUMMARY

1. We have presented data from 160 successive gynecologic operations, wherein cotton suture material has been employed exclusively.

2. Results obtained have more than justified the use of this material as indicated by wound healing, cosmetic results, and postoperative comfort, all largely explained by lack of tissue reaction.

3. We have not felt handicapped by working with cotton as shown by the high incidence of complete hysterectomy (94 per cent) as opposed to supracervical hysterectomy (6 per cent).

4. We recommend the wider use of cotton suture material in gynecology, following the tenets of Halsted, and suggest early ambulation of postoperative patients in the absence of any distinct contraindication.

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INTRATHORACIC ESOPHAGOJEJUNOSTOMY FOR TOTAL GASTRECTOMY WITH LOWER ESOPHAGECTOMY FOR CARCINOMA

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TOTAL gastrectomy has become a more common procedure in recent years. The morbidity and mortality statistics of this operation are also improving steadily. Therefore, it seems advisable to attempt this procedure more often in indicated cases.

Similarly, operations for carcinoma of the cardiac end of the stomach and lower esophagus are being successfully developed. Larger series of intrathoracic esophagogastrostomies are reported.

At the recent meeting of the American Association for Thoracic Surgery held in Toronto, in June, 1941, several papers were read on carcinoma of the esophagus. Wookey, of Toronto, presented cases of successful operations for carcinoma of the cervical portion of the esophagus.

Hence, all the portions of the esophagus and stomach are now becoming more amenable to radical surgical resection.

Recently we had occasion to perform a total removal of the stomach together with the lower two inches of the esophagus, both of which were involved in a linitis plastica type of carcinoma. Reconstruction was accomplished by an intrathoracic esophagojejunostomy.

The patient survived the operation. Death, five months after the operation, was due to general carcinomatosis. Various lessons were learned from this case and therefore it seems worth while to publish these experiences, hoping it may encourage others to attempt the same procedure. Progress will only come in this field if we learn and profit by the experiences of others.

After a careful study of the published literature, it has been found that this case is apparently the first one of a successful intrathoracic esophagojejunostomy. Churchill, of Boston, reported a somewhat similar operation during the discussion at the Toronto meeting of the American

This case was presented before the joint meeting of the Philadelphia Academy of Surgery and the New York Surgical Society on Feb. 12, 1941. It was also reported at the Toronto meeting of the American Association for Thoracic Surgery.

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Association for Thoracic Surgery. His patient, unfortunately, did not survive the operation.*

CASE REPORT

L.D., a 52-year-old Swedish woman, was admitted by me to the Lenox Hill Hospital on Sept. 28, 1940. She had a history of two and one-half years of pain in the gastric region. Two years before admission she had gone to a doctor. X-rays were taken and reported negative (Fig. 1), but it was suggested that they be repeated. Powders and diet gave relief. There followed a several months' period of remission of symptoms. The patient did not return to the doctor. Ten months before admission the symptoms returned and this time were not relieved by treatment. Nausea and vomiting were present. There was marked loss of appetite and loss of forty pounds in weight since the onset of the disease.

The past history and family history were irrelevant. The patient was married and had three children.

Physical Examination.—Head and neck were negative. Heart and lungs were negative. In the abdomen there was some fullness in the epigastrium but no definite tumor was palpable. Weight, 130 pounds; pulse, 68; blood pressure 108/65.

Laboratory Findings.—Gastric analysis following a test meal showed absence of free hydrochloric acid and a low total acidity of 10. Lactic acid and blood were absent. Wassermann was negative. Blood count, Hb., 92 per cent; red blood cells, 4,750,000; white blood cells, 11,600; polymorphonuclear leucocytes, 64 per cent; lymphocytes, 33 per cent; monocytes, 1 per cent; eosinophiles, 2 per cent.

X-ray Examination.—Following a barium meal no obstruction or deformity of the esophagus was seen at fluoroscopy. A flat, filling defect measuring approximately 7 cm. in length was seen in the region of the midgreater curvature extending to the posterior wall of the stomach. This portion of the stomach seemed markedly stiffened and did not transmit peristaltic waves. Slight irregularity of the upper greater curvature was noted, which may possibly represent further extension toward the fundus. The prepyloric region and duodenum appeared normal (Fig. 2). At six hours the stomach was empty except for traces of barium. The deformity in the middle third of the stomach had the characteristic roentgen appearance of a malignant neoplasm.

Permission was given by the patient and the family for an exploratory operation and it was understood that a total gastrectomy might be necessary. A transfusion of whole blood of 500 c.c. was given two days before the exploratory operation.

First Operation.—On Oct. 2, 1940, under general anesthesia, a left paramedial incision was made. A tremendous tumor of the stomach was found involving the middle third and entire fundus of the stomach. The gastrohepatic omentum was split and exploration showed a firm hard infiltration around the gastric vessels in

*The operation performed for a similar lesion was done April 2, 1941. It was an extensive carcinoma involving the lower third of the esophagus extending 2½ cm. above the diaphragm to the midportion of the body of the stomach. The entire procedure was performed through a thoracic approach with resection of the tenth rib. The entire stomach was resected, together with the tips of the pancreas and spleen, and the portion of the esophagus involved in the tumor. An intrathoracic esophagojejunostomy was performed with some difficulty due to the edema of the esophagus and the cutting through of the sutures. The anastomosis was not completely satisfactory for this reason. An enteroenterostomy was performed just below the opening in the transverse mesocolon where the loop of the jejunum was brought up. A leaf of omentum was brought up to cover the jejunum and the region of the anastomosis, and the diaphragm was reapproximated in two layers around it. Airtight drainage was placed into the pleural cavity. The patient stood the operation well but then ran a course of continued sepsis with high fever and died on the ninth postoperative day. A post-mortem examination was obtained but limited to the region of the operation. There was evidence of a defect along the suture line of the anastomosis with resulting empyema and a localized abscess under the diaphragm extending from the surgical hiatus of the diaphragm posteriorly to involve the end of the pancreas and the duodenal stump. The enteroenterostomy was water tight and the suture line of the duodenal stump was intact.

the region of the celiac axis. The upper limit of the tumor could not be felt at the cardia. Therefore the lateral ligament of the left lobe of the liver was divided and the lobe displaced toward the right exposing the esophageal hiatus of the diaphragm. The peritoneum was incised and the esophagus isolated and surrounded with a tape. The esophagus was found to be involved in a continuous mass with the stomach. The tumor extended up the wall of the esophagus into the posterior mediastinum for a distance of about 5 cm. Above this, smooth and soft pliable esophageal wall could be palpated but not visualized. The entire tumor mass was movable everywhere except possibly in the region of the gastric vessels posteriorly.

Fig. 1.—X-ray taken in May, 1939, at which time carcinoma of the stomach was not recognized.

There were no palpable metastases in the pelvic shelf and no palpable liver metastases. Several lymph nodes along the greater curvature were enlarged and several were removed for pathologic examination. It was realized that radical removal of the entire tumor mass was possible only by performing a total gastrectomy with a combined laparotomy and transthoracic resection of the lower esophagus with splitting of the diaphragm and then intrathoracic esophagojejunostomy. This procedure is of such magnitude that it did not seem wise to attempt it at this time without special preparation of the patient and full consent of the family. Therefore nothing

further was done and the abdomen was closed in layers. The patient made an uneventful recovery. Pathologic examination of the removed lymph nodes showed only hyperplasia.

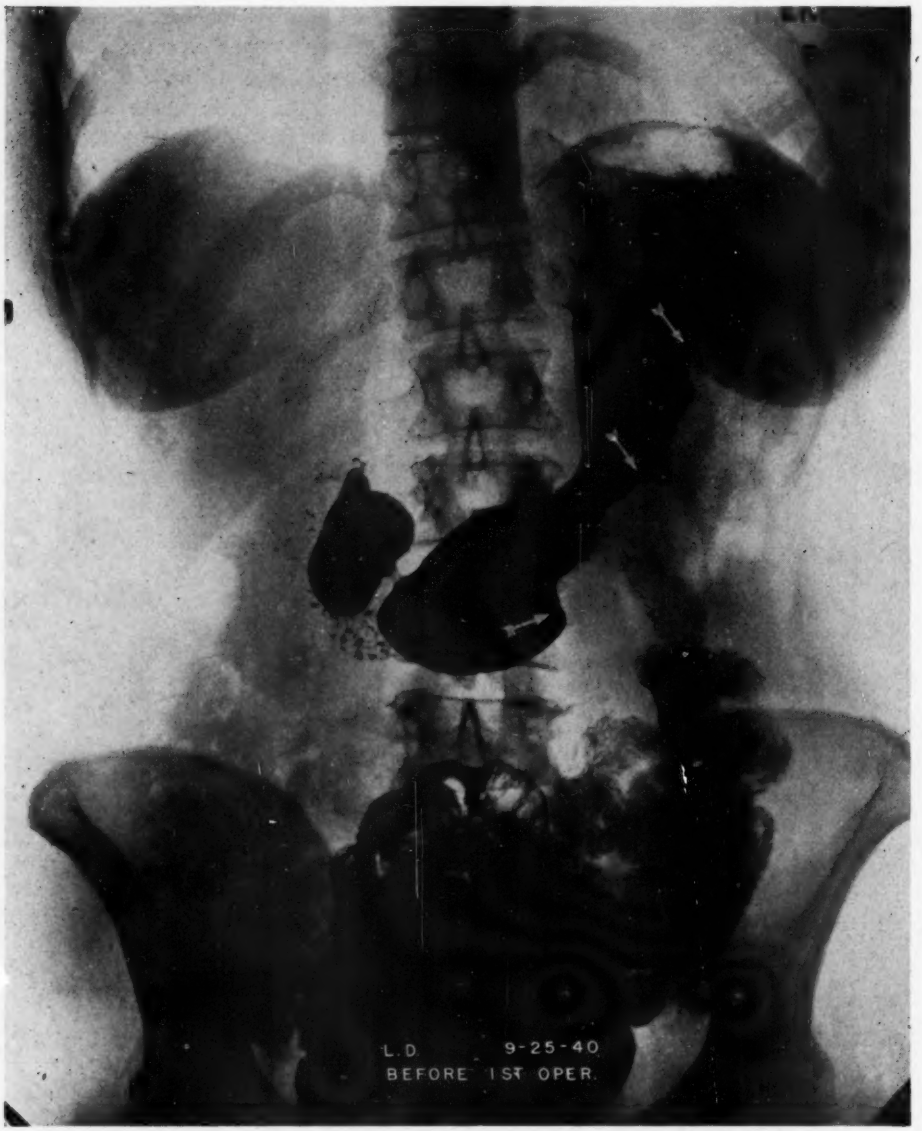


Fig. 2.—X-ray taken before operation showing extensive involvement by carcinoma.

Interim Course.—With the full knowledge of the extent of the lesion an esophagoscopy was performed by Oberrender on the twenty-sixth postoperative day. The findings were as follows: 36 cm. from the upper jaw the esophagus was constricted and the mucosa was thickened but not irregular. This occurs in a concentric fashion and gives the impression of being a periesophageal infiltration with no signs whatever of neoplasm within the lumen. Biopsy was taken from the posterior wall where the thickening of the mucosa was most marked. Pathologic examination of this specimen showed edematous esophageal mucosa with no sign of malignancy. X-ray

examination made at this time showed no constriction or deformity of the esophagus except little, if any, dilatation of the esophagus above a slight constriction at the lower end (Fig. 3). The patient was discharged from the hospital, Nov. 4, 1940, the thirty-third postoperative day, for a short period of home convalescence and preparation for the second operation. Consent for this procedure as outlined above was given by the family. Weight on discharge was 120 pounds.

While at home the patient was on a high protein and caloric diet with accessory vitamins, especially cevitamic acid and vitamin B complex. After twelve days she was readmitted to the hospital with a gain in weight of two pounds.

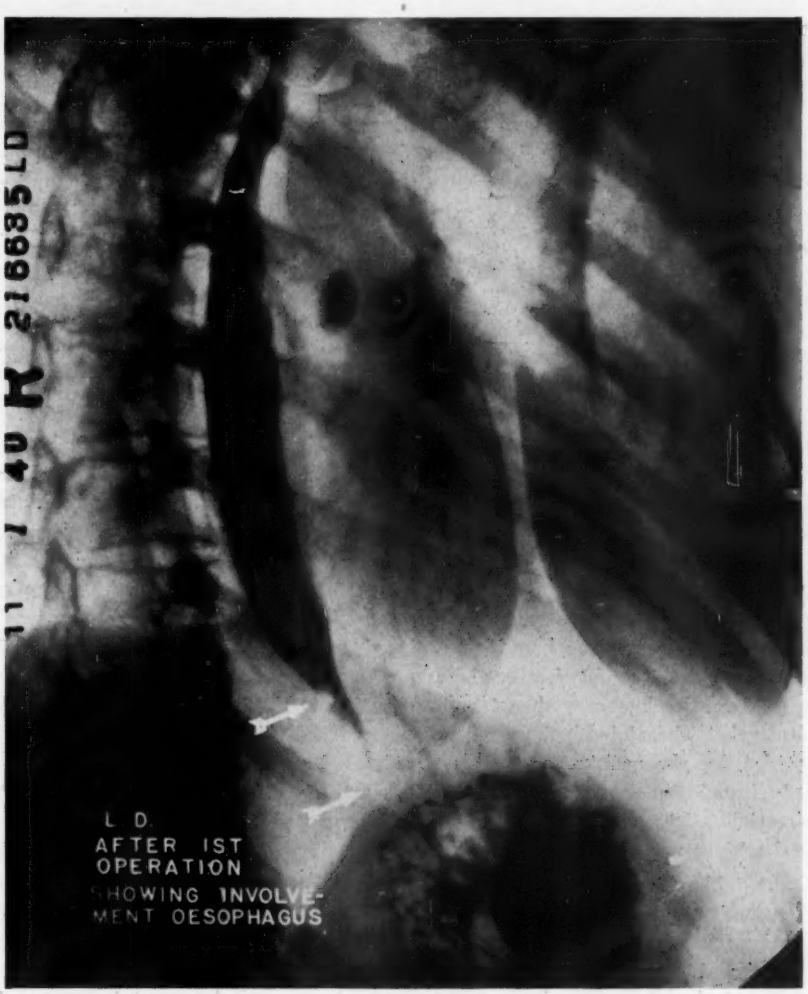


Fig. 3.—X-ray of lower end of esophagus taken after the first operation with knowledge of presence of carcinoma of lower end of esophagus. Narrowing of lumen of esophagus is shown.

Preoperative preparation was as follows: (1) High protein diet; (2) cevitamic acid and vitamin B complex; (3) daily intravenous infusions of 1,500 c.c. of 5 per cent glucose in saline solution for three days before operation; (4) blood transfusion of 400 c.c. of blood two days before operation; (5) sulfanilamide gr. 15 every four hours for three days before operation; (6) collection of 2,000 c.c. of citrated blood the day before operation.

Clinical Findings Before Second Operation.—Two days before operation the blood count was as follows: Hemoglobin, 12 Gm. or 83 per cent; red blood cells, 3,850,000; white blood cells, 10,800; polymorphonuclear leucocytes, 65 per cent; lymphocytes, 30 per cent; monocytes, 2 per cent; eosinophiles, 3 per cent. Red cell volume, 44 per cent; blood plasma specific gravity, 1.0256; blood plasma protein, 6.36 per cent; blood plasma chloride, 616 mg./100 mil. Total base, 150 meq. per liter.

Second Operation (Nov. 22, 1940).—Basal avertin with inhalation anesthesia was used. Intravenous infusion was started before operation by placing a cannula into the vein of the left arm as all the superficial veins were so small and invisible that this was the only feasible method. A Levin tube was placed into the stomach to keep this empty.

The scar of the previous laparotomy just to the left of the midline was excised. The abdomen was opened and recent adhesions between the abdominal wall, omentum, stomach, and left lobe of the liver were freed. The gastrocolic omentum was divided from the pylorus along the greater curvature to the gastrosplenic ligament and along the fundus. The gastrohepatic omentum was then divided from the pylorus to the region of the gastric artery. The duodenum was divided with the aid of the dePetz clamp, the stump buried with silk sutures, and the head of the pancreas stitched over the suture line. The stomach was then easily collapsed by suction on the indwelling Levine tube and the further freeing of the stomach continued. The stomach was adherent to the under surface of the diaphragm in the region of the esophageal hiatus where the peritoneum had been incised at the time of the first operation. These adhesions were severed between ligatures with some difficulty. The greatest difficulty was to free the stomach posteriorly in the region of the gastric artery. This division was finally accomplished, freeing the entire stomach up to the esophagus at the diaphragm. The esophagus was then freed at the hiatus as far as the finger could reach. A laparotomy pad was wrapped around the stomach and tied with tape. Hereupon a loop of jejunum was taken about eighteen inches from the duodenojejunal juncture and brought upwards toward the diaphragm antecolically. At the lower end of these two limbs an enteroenterostomy was performed. One black silk thread was placed as a marker near the dome of the afferent loop and two black silk threads as markers near the dome of the efferent loop. This entire loop was then temporarily tied to the laparotomy pad surrounding the stomach. The abdominal wound was then closed using through-and-through steel alloy wire for the peritoneum, rectus muscle, and rectus sheath. Several fine interrupted chromic sutures were placed between these steel alloy wires for exact approximation. The skin was closed with interrupted silk sutures and the wound dressed with gauze and adhesive.

Citrated blood was started at this time through the cannula in the brachial vein. The patient was then placed on her right side. An intercostal incision was made along the seventh interspace, a small section of the seventh, sixth, and fifth ribs being resected near the vertebral column. The intercostal tissues were ligated and divided and the pleural cavity entered along the seventh intercostal space and the incision lengthened upward posteriorly. The rib spreader was inserted. Adhesions between the lung, parietal pleura, and upper surface of the diaphragm were transilluminated and then divided, freeing the lung, the upper surface of the diaphragm, and the mediastinum. The pleura over the mediastinum was incised exposing the esophagus all the way down to the diaphragmatic hiatus. The upper end of the tumor was felt about 5 cm. above the diaphragm. The phrenic nerve was crushed, paralyzing the diaphragm. The latter was then incised from the central tendon to the hiatus, making it very easy to draw up the stomach, with jejunal loop attached, into the thoracic cavity. It was at once disappointing to note that the mesentery

of the jejunal loop was not long enough to bring this up into the chest as easily as had been anticipated. The Levin tube was then withdrawn out of the stomach into the esophagus just above the level of the tumor. Continuous suction was applied to the tube to keep the esophagus empty. The entire mediastinum and lower chest cavity was protected with gauze pads moistened in one-half per cent lysol solution in order to try to prevent gross soiling of these areas. The efferent loop, marked with two black silk threads, was then sutured to the posterior surface of the esophagus at a point above the upper limit of the tumor. There was a good deal of tension and in order to relieve this the jejunum was temporarily sutured to the cut edge of the diaphragm. After the esophagus had been fastened thus to the jejunum posteriorly the esophagus was divided above the tumor and the specimen composed of the entire stomach and lower 5 cm. of the esophagus was removed in one piece (Fig. 4*A* and *B*). Hereupon an incision was made into the jejunum just distal

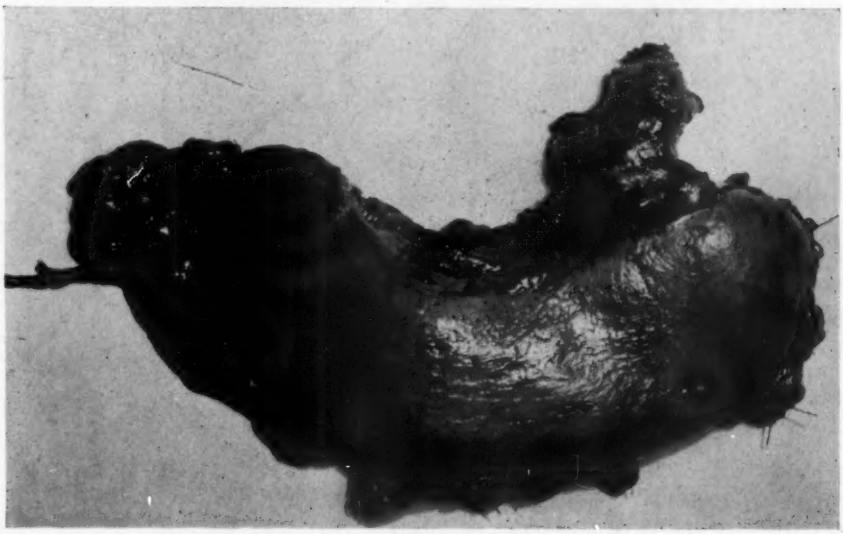


Fig. 4*A*.—Photograph of specimen of stomach including the lower 5 cm. of the esophagus.

to the dome of the jejunal loop. The Levin tube which had been drawn into the esophagus was then pushed down again and into the efferent loop of the jejunum for as long a distance as possible. There was some difficulty in doing this and it could not be pushed down as far as had been hoped. Esophagojejunostomy was then completed. It was necessary to place interrupted mattress silk sutures to successfully close the anterior suture line because straight sutures easily cut through the friable esophagus due to the unfortunate tension. After the anterior suture was placed, the afferent loop of the jejunum was brought over across the suture line in a manner similar to the technique proposed by Roscoe Graham in the intra-abdominal esophagojejunostomy in total gastrectomy. It was not possible to do this in an ideal manner but a covering had to be improvised and the parietal mediastinal pleura was also used to cover and reinforce the anastomosis. The jejunum which had temporarily been sutured to the cut surface of the diaphragm to relieve tension during the performance of the anastomosis was then separated and resutured in the proper position close to the esophageal hiatus in the diaphragm. The diaphragm was then sutured from the central tendon toward the jejunum at the hiatus. The esophagojejunostomy then looked quite satisfactory although there was definitely more tension than had been anticipated. Intercostal airtight drainage

was instituted two interspaces below the incision and the drainage tube placed fairly close to the region of the anastomosis. The chest was closed in the usual manner using chromic catgut around the ribs, continuous fine chromic catgut for the intercostal muscles and for the muscles, giving airtight closure. The lung was expanded during this procedure. The skin was closed with interrupted silk sutures and compression dressing applied.

Fig. 4*B*.—Photograph of specimen opened showing the thickened walls of the esophagus and stomach containing the carcinoma.

The patient's general condition was satisfactory at the end of the operation. The pulse was of fair quality, the rate 120, and the blood pressure satisfactory.*

Postoperative Course.—The patient was placed immediately into an oxygen tent and the blood transfusion continued through the cannula. Following the transfusion 5 per cent glucose in saline solution was substituted alternating each 1,000 c.c. between saline solution and distilled water. The patient was stimulated alternately with metrazol and caffeine sodium benzoate. During the first day there was 400 c.c. of pleural drainage. On the second day 500 c.c. of citrated blood was given through the intravenous cannula. Some water was allowed to flow by gravity through the indwelling Levin tube; this worked well. On the third day 10 per cent glucose in saline solution was allowed to drip through the Levin tube at the rate of 125 c.c. per hour. The infusion was continued. Blood hematocrit on this day was normal, as were the chlorides and serum protein. The infusion was discontinued, therefore, after having run continuously for three days. Albumin water was added through the Levin tube and on the fourth day ice pills were allowed by mouth. One-third milk and lime water with cevitamic acid was given through the tube. On the fifth

*I am indebted to Dr. Herbert C. Maier for his assistance during this operation.

day one-half ounce of water was given every hour by mouth. On the following day there was intestinal content drainage around the pleural drainage tube with profuse drainage through the tube itself. Methylene blue and milk swallowed by mouth did not appear through the drainage tube at this time. With this leakage of intestinal content fluid balance was maintained easily through the Levin tube. Temperature varied between 99.6° and 101.6° F. since the operation. There was no evidence of spreading mediastinitis. Bedside x-ray examination showed the lung well expanded. On the tenth day both wounds were dressed. They were clean and healed by primary union and all sutures were removed. The pleural drainage tube was changed and a transfusion of 500 c.c. of whole blood given. On the nineteenth day x-ray examination with barium swallowed showed that most of it ran into the jejunum. On the twenty-first day the intestinal fistula was closed and all feedings were given by mouth. On the twenty-second day the chest drainage tube was shortened and on the twenty-fourth day the Levin tube was withdrawn easily and the patient

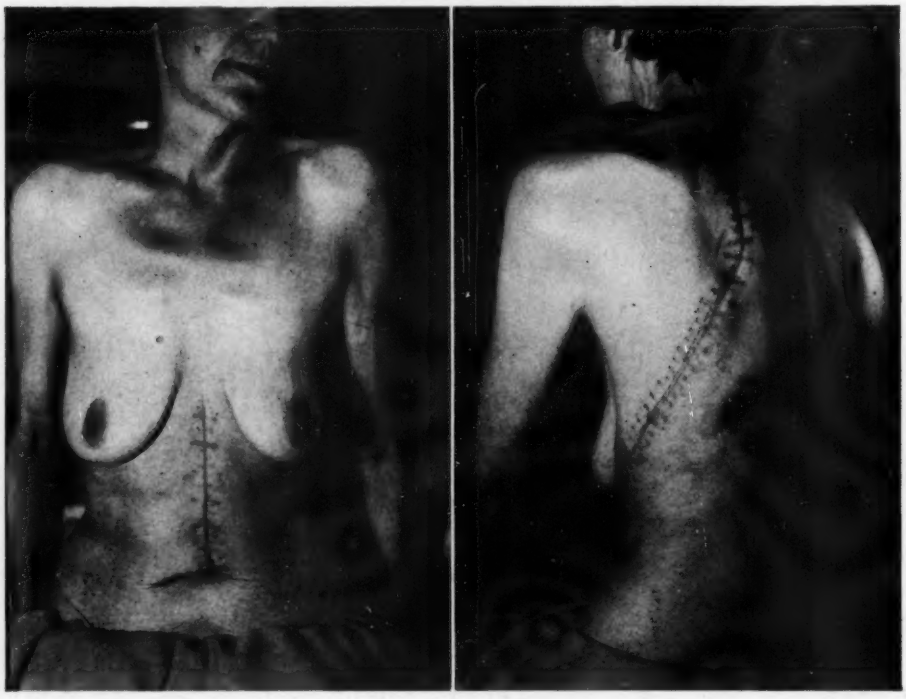
Fig. 5.—Low-power microphotograph showing involvement of submucosa of esophagus by carcinoma.

allowed out of bed. The fistula had lasted nineteen days. On the thirty-second day the chest tube was withdrawn and two days later the patient was receiving 2,100 calories per day by mouth with additional vitamins in liver extract. The patient's weight had dropped to a low of 102 pounds (Fig. 6*A* and *B*).

Postoperative Clinical Findings.—Blood examinations six weeks after operation. On Jan. 4, 1941, the hemoglobin was 9.6 Gm. or 63 per cent; red blood cells, 3,030,000; white blood cells, 10,700; polymorphonuclears, 73 per cent (28); lymphocytes, 26 per cent; monocytes, 1 per cent. Blood urea nitrogen, 7.2 mg. per 100 c.c.; creatinine, 0.5 mg.; uric acid, 2.4 mg.; sugar, 76 mg.; CO_2 combining power, 51.9 per cent. Hematocrit, 38; plasma specific gravity, 1.0244; plasma protein, 5.54; plasma albumin, 3.34; plasma globulin, 2.20; plasma chlorides, 549; serum diastase, 76. One month later, Feb. 7, 1941, the values of blood examinations were: 12.2 Gm. hemoglobin; red blood cells, 4,050,000; white blood cells, 9,200; polymorphonuclear leucocytes, 72 per cent (24); lymphocytes, 24 per cent; monocytes, 2 per cent; eosinophiles,

2 per cent. Hematocrit, 45; blood plasma, specific gravity 1.0247; plasma protein, 6.05; plasma CO_2 combining power, 68.3; plasma chlorides, 546; total base, 145.0, and serum diastase, 41. Stool examination, Jan. 4, 1941, revealed many yeast cells, some digested and undigested muscle fibers, some vegetable remains, occasional white blood cells, occasional fat globules, and bacteria.

Pathologic Examination.—The specimen received fresh was a stomach complete with 5 cm. of the esophagus attached. The stomach measured 17 cm. in length, 7 cm. in width, and 3.5 cm. in thickness. The serosal surface was tan-pink, smooth and glistening throughout. The wall over the entire stomach including the first 2 to 3 cm. of the esophagus was markedly thickened and firm. This thickened, firm area ended about 4 cm. from the duodenum. The rugae were markedly elevated. The mucosal surface was mottled, tan-pink to purple in color. On section, the wall throughout the thickened area was gray-pink, firm, smooth, and glistening.



A. *B.*

Fig. 6.—*A*, Photograph of patient after operation showing healed laparotomy wound. *B*, Photograph of patient showing healed thoracotomy wound.

Microscopic examination of sections taken through the thickened area revealed a tumor arising in the mucosa of the stomach. It replaced the mucosa in a small area, but infiltrated the wall of the stomach throughout. The tumor cells were small or medium sized with a scanty cytoplasm and fairly large, vesicular or hypochromatic nuclei. Mitotic figures were inconspicuous. These cells formed no regular structures, being seen often singly or in groups, though occasionally they formed small nests and cords which infiltrated between the muscle fibers. They were supported and accompanied by a rather abundant connective tissue stroma which was quite cellular and markedly infiltrated with round cells. The growth invaded all the

layers of the stomach wall and extended beneath the intact mucosa into the esophagus to within 5 mm. from the edge of the specimen (Fig. 5). The pyloric end of the stomach was free of tumor tissue. The diagnosis was carcinoma of the stomach (linitis plastica).

Final Postoperative Course.—The remainder of the patient's course was a gradual decline. The esophagojejunostomy anastomosis was easily dilated several times with an olive bougie. Swallowing was satisfactory for the remainder of the patient's life (Fig. 7). The appetite steadily declined and carcinoma cachexia set in. Shortly before the death of the patient a jejunostomy was performed (March 30,

Fig. 7.—X-ray showing functioning of anastomosis four months after operation. Note level of diaphragm and loop of jejunum above level of diaphragm within thoracic cavity. Loop is somewhat dilated to take the place of the stomach. Wire staples in the duodenal stump and steel alloy wires in the abdominal wall are also to be seen.

1941) for feeding purposes. At the time of this operation it was seen that the patient had a generalized carcinomatosis involving the liver, parietal and visceral peritoneum, intestinal mesentery and omentum. X-ray examination of the chest showed evidence of pulmonary metastases. The patient died on April 14, 1941, approximately five months after the major operative procedure. Unfortunately, an autopsy could not be obtained.

COMMENT

Total gastrectomy with removal of the lower esophagus and an intrathoracic esophagojejunostomy is feasible. This, I think, is best performed by means of a laparotomy and immediately following thoracotomy. Experience in this case shows that it is better to bring the loop of the jejunum up into the thorax retrocolically instead of antecolically. Examination on cadavers at the autopsy table since this experience, has shown that the entire root of the mesentery slides upward through the retrocolic opening in the transverse mesocolon for a small distance and therefore allows the jejunum to be brought up higher into the chest with resulting lessened tension on the suture line at the site of the anastomosis. The enteroenterostomy which is, of course, essential should lie then just below the opening in the transverse mesocolon.

If difficulty is found in freeing the stomach in the region of the spleen Lahey has recently advised the removal of the spleen together with the stomach rather than spending time in trying to separate the stomach from the spleen.

A jejunostomy performed at the same time as the laparotomy (Clute and Bell) is definitely better than the indwelling Levin tube passing through the site of anastomosis. This tube at the site of the anastomosis probably tends more to the development of a fistula with leakage. In a recent successful total gastrectomy, we have employed a jejunostomy for feeding purposes with great satisfaction. It adds very little time to the procedure, being accomplished in a few minutes. It has been a question in my mind whether the intestinal fistula in this case originated from an area of necrosis or damage to the intestinal wall at the point where the jejunum had been sutured temporarily to the edge of the diaphragm. On the other hand, it is more likely that it came from the site of the esophagojejunostomy anastomosis. However, it was noteworthy that methylene blue solution and milk did not immediately appear from the fistula opening but the drainage was mostly intestinal in character. At any rate the fistula lasted nineteen days and then closed spontaneously.

The preoperative preparation of these patients is of vital importance in all cases of major, upper abdominal surgery. High protein diet and high vitamin intake are essential. Vitamins A and D are important for healing of epithelial structures. Vitamin B complex is important for the proper functioning of the gastrointestinal tract and vitamin C is important for the intercellular cement and the prevention of edema at the site of the anastomosis.

The formula for jejunostomy feedings as suggested by Clute and Bell is very valuable and we have been using it to advantage. It is as follows: peptonized skimmed milk, 500 c.c.; one egg, well-beaten; haliver oil

and viosterol, 3 minims; syrup of vitamin B complex, 15 c.c.; cevitamic acid, 300 mg.; thiamine chloride, 6 mg.

Blood transfusion before operation and the use of citrated blood or blood plasma during the operation are most valuable adjuvants.

Sulfanilamide or sulfadiazene before operation helps to prevent infection below the diaphragm and within the pleural cavity and mediastinum. Recently, in a case of total gastrectomy I used 4 Gm. of sulfanilamide powder under the diaphragm at the site of the anastomosis. In the future I will use sulfanilamide powder in the posterior mediastinum and pleural cavity in place of the lysol wipes employed in this case.

An oxygen tent postoperatively is a great help for the patient. It maintains a high oxygen concentration and keeps the patient in a steady cool temperature which relieves breathing a great deal.

SUMMARY

1. A successful case of total resection of the stomach with the lower 5 cm. of the esophagus is reported. It was followed by an intrathoracic esophagojejunostomy.

2. Preoperative preparation, operative technique, and postoperative care are discussed. These factors are most important for success in such major procedures.

3. It is hoped that the report of this case, though fatal five months after the operation due to generalized carcinomatosis, may encourage other surgeons to attempt similar operations. The experience of others will help to improve the technical procedure. Only by experience, and its report in literature, can better results be obtained.

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AN OPERATION FOR INGUINAL HERNIA BASED UPON THE UTILIZATION OF COOPER'S LIGAMENT*

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THE numerous modifications of the classic Bassini operation for inguinal hernia imply clearly that the original operation has not proved entirely satisfactory. The sole justification for an addition to the long list lies in the belief that the proposed operation offers direct support, as well as some measure of reduction, of the undefended space. The Bassini operation and its variations all lay down a shelf which is intended to resist permanently a hernial protrusion. Whether the conjoined tendon or rectus sheath or aponeurosis of the external oblique are joined to the reflected surface of Poupart's ligament, with or without re-enforcement by other tissues (fascial sheets or sutures), the objective remains a protective shelf and not a direct support of the undefended space. The latter may be partially reduced by closure of the gap in the transversalis fascia. Essentially, however, complete reliance against the recurrence of a hernial protrusion is placed on the barrier of a shelf which is not directly applied to the undefended space. The following operative procedure offers direct support to the mesial portion of the undefended space in the form of a slinglike buttress, and also reduces to some degree the size of that space. Cooper's ligament is utilized for the purpose, Poupart's ligament being relied upon for support in the conventional manner.

Technique: The cutaneous incision is placed somewhat nearer the inguinal fold than usual and the external oblique is split lower than usual in order to fashion a generous flap of the aponeurosis. A broader flap of external oblique than usual is required, for its approximation to the deeply situated Cooper's ligament is the essential part of the operation. After the isolation of the structures of the inguinal canal and the customary ablation of the sac (in the case of indirect inguinal hernia), the direct bulge or hernia is depressed with a suitable instrument. If there is serviceable transversalis fascia for the purpose, a purse-string suture is passed and tied for the inversion of the direct hernia. The essential step is the utilization of Cooper's ligament. The latter is exposed close to the spine of the pubis by following the reflected (deep) surface of Poupart's ligament into the pelvis. A thin but definite fibrous

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membrane medial to the iliac vein and apparently continuous with Poupart's ligament is traversed, the blunt dissector used for the purpose following the curve of the pelvis. The glistening free surface of Cooper's ligament, closely applied to the pubic ramus, is visualized by the placement of a narrow (one-quarter inch) malleable (ribbon) retractor. The external iliac vein is sufficiently isolated for its gentle lateral retraction with a second ribbon retractor, and Cooper's ligament is thereby fully exposed.

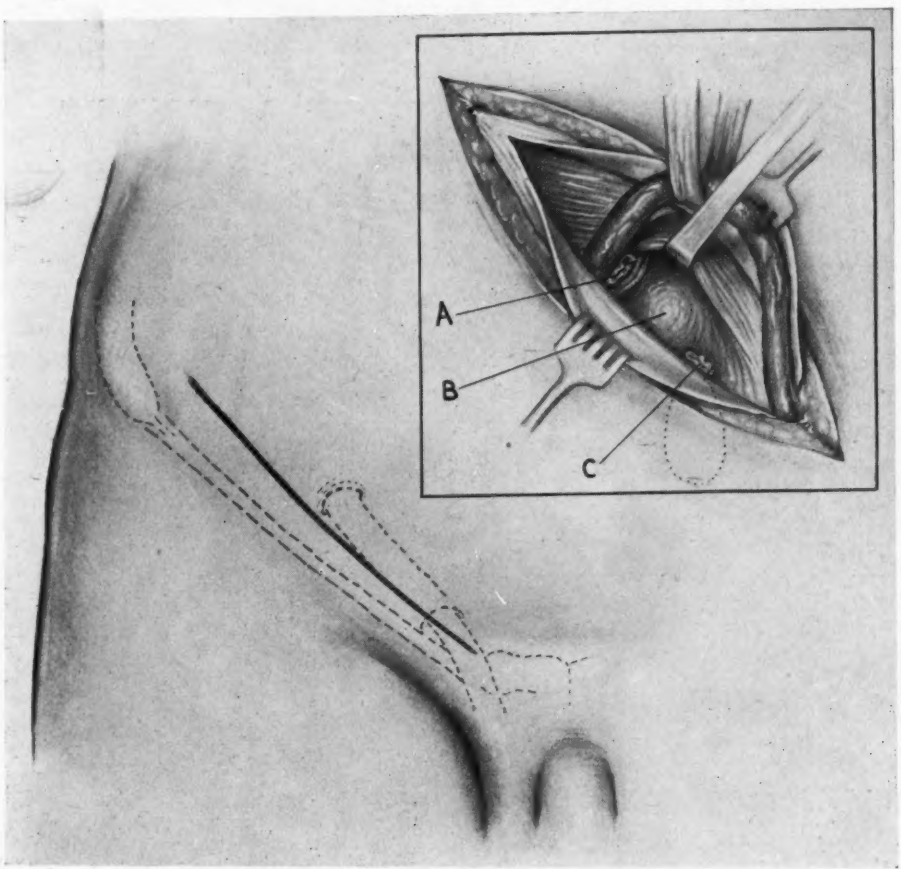

Fig. 1.—Showing that the inguinal incision is lower than usual. In the insert are indicated (*A*) the stump of an ablated indirect sac, and (*B*) the protrusion of a direct hernia. If operation for femoral hernia is performed through an inguinal incision, the situation of the stump of an ablated femoral sac is indicated (*C*).

Two (or three) sutures, closely hugging the periosteum, are now passed through Cooper's ligament. I have found that the use of the Reverdin needle facilitates the passage of these deep sutures. In any event, attention should be called to the fact that the sutures are not correctly placed unless they are closely applied to the bone. After the passage of the sutures, which may be attended with some difficulty because of depth, the two ribbon retractors are removed. The mobilized aponeurosis of the external oblique is now drawn downward with two forceps and the

two (or three) sutures are passed through it at appropriate sites. The approximation should be tested before the sutures are tied. When tied, the external oblique should be drawn without undue tension downward and into the pelvis in the form of a sling. This curved support extending into the pelvis snugly supports and reduces appreciably the undefended space medial to the iliac vessels. If the sutures have been placed correctly, a pronounced curvature of the external oblique aponeurosis should be evident, and no dead space should remain. Poupart's ligament is now utilized. Its reflected surface lateral to the sling is approximated

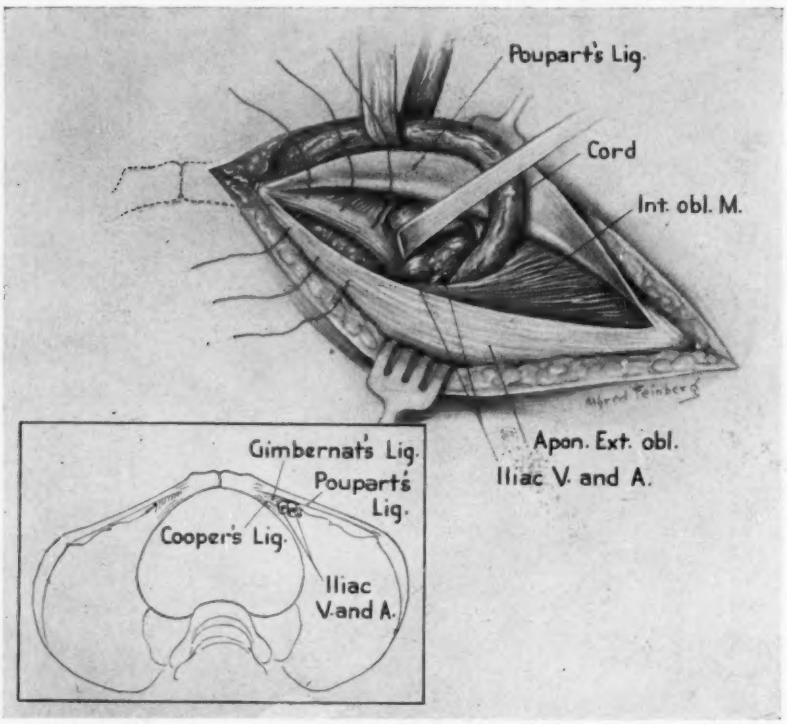

Fig. 2.—Sutures are passed between the aponeurosis of the external oblique and Cooper's ligament. Some of the fibers of Gimbernat's ligament may be included with advantage. The iliac vessels are not isolated as shown in the drawing.

to the external oblique in the customary manner. The medial portion of the reflected surface is then approximated by a few sutures to the surface of the external oblique over the sling in order to provide additional support. Silk technique is advocated.

The foregoing operative procedure has been employed for about three years. At first applied only to selected cases of direct inguinal hernia, it is now utilized for all direct hernias as well as for indirect hernias in which a direct component exists. There appears to be no reason to omit the procedure whenever the requirement is a direct support of the mesial portion of the undefended space.

There have been no accidents referable to the operative procedure and none need be anticipated with careful technique. All structures in the field should be clearly visualized with special reference to the iliac vein. The sole danger of operation is damage to the vein, an accident which is obviated by gentle lateral retraction of that structure.

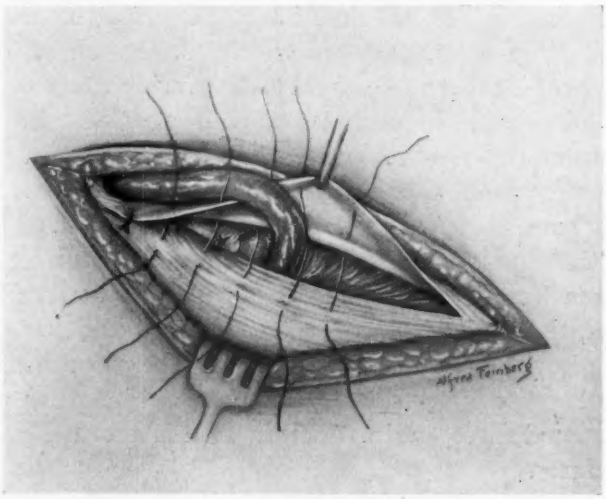

Fig. 3.—The sutures through Cooper's ligament have been tied and the reflected surface of Poupart's ligament is approximated to the adjacent aponeurosis of the external oblique. The structures of the cord can be displaced laterally to a greater extent than illustrated in the drawing.

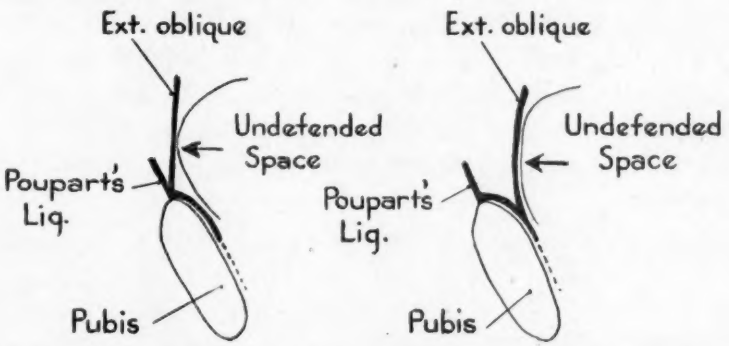

Fig. 4.—Vertical sections to illustrate the difference in effect between the Bassini type of operation and that described in the text. Left: The external oblique is attached to the reflected surface of Poupart's ligament and the undefended space is not directly supported. Right: The external oblique is attached to Cooper's ligament, Poupart's ligament not being utilized in the first layer. The undefended space is directly buttressed.

As compared with the usual operative exposure for hernioplasty, the field will appear surprisingly deep the first few times the described operation is performed. As a matter of fact, the depth always is great when the operation is performed for direct hernia in a stout individual. An unusually deep situation of Cooper's ligament comprises the only difficulty in the operation. As has already been stated, the passage of the suture through Cooper's ligament is facilitated by the use of the

Reverdin needle. Ordinary needles of small size can, however, be employed satisfactorily if their passage is not hurried. It is sometimes more convenient to pass the sutures through Cooper's ligament from the opposite side of the wound (side of the first assistant). If the sutures have been correctly placed, the aponeurosis of the external oblique can be approximated to Cooper's ligament without undue tension. After the sutures are tied, the result must be a firm, incurved, slinglike buttress and not a shelf. Of course, the utilization of the reflected (deep) surface of Poupart's ligament as an overlap offers additional firm support but this should be regarded as rather incidental insofar as the mesial portion of the wound is concerned. The utilization of Poupart's ligament as the essential structure for support begins immediately lateral to the sutures passed through Cooper's ligament.

The fact that uniformly good results in cases of direct and direct-indirect hernia have followed the described operation has little significance because the number of cases is too small and the time elapsed since the operation has been practiced is too short. The procedure is advocated at this time because it appears to offer a more definitive assurance of cure of direct and direct-indirect hernia than is afforded by the conventional procedures.

A RECONSTRUCTION OPERATION FOR LARGE MIDLINE INCISIONAL HERNIA

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THERE are several satisfactory methods for the repair of midline incisional hernia. It is for the extremely large, neglected hernia with wide separation of the anterior abdominal wall that we wish to describe this technique.

The Deformity.—(Fig. 1.) The hernial sac, by its mushroom shape, frequently gives the impression of a much larger opening than is actually found at operation. Since most pelvic operations are carried out through a right paramedian incision, the left rectus muscle is usually found to be completely inclosed in a normal sheath. The right rectus muscle frequently gives a similar impression, but actually the fascia of the anterior sheath is apt to be found retracted some distance from the hernial margin. The peritoneum, in providing the lining for the hernial cavity, is firmly adherent to the medial border of the muscle and the fascia of the anterior sheath by rather dense scar tissue simulating, in many instances, true fascia. Except, then, for the fact that there is an actual break in the continuity of the muscular and fascial structures of the abdominal wall in the midline, and that a hernial sac is present, we have a mechanical problem here differing not greatly from that of diastasis recti in this portion of the abdominal wall.

The method of reconstruction, here described, is an adaptation of that originally used and later described by us[1] for the repair of diastasis of the rectus muscles. This principle has been used in a fairly large series of cases over the past several years with almost uniformly good results. Beef fascia, it will be noted, is used for the muscular support instead of fascia lata. Clinically, beef fascia has been found to be perfectly satisfactory for this purpose, and it has been used by us over a period of fifteen years.

TECHNIQUE OF OPERATION

The Dissection.—(Figs. 2 and 3.) A large midline incision is made and the hernial sac is freely exposed. The sac is opened in its midportion. The lateral walls are dissected free from the overlying skin and fat. The dissection is carried laterally thoroughly to expose the

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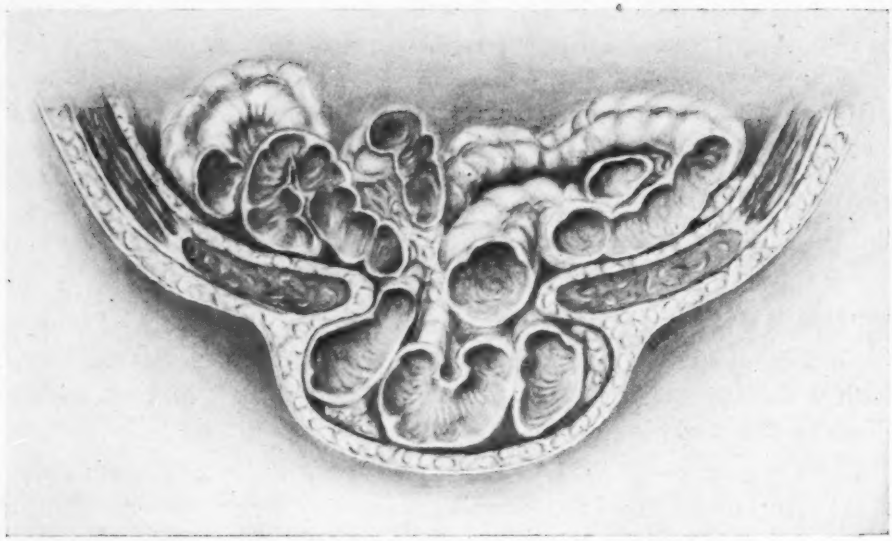


Fig. 1.—Cross section through hernia. Note the separation of the rectus muscles. The circular muscles are shortened.

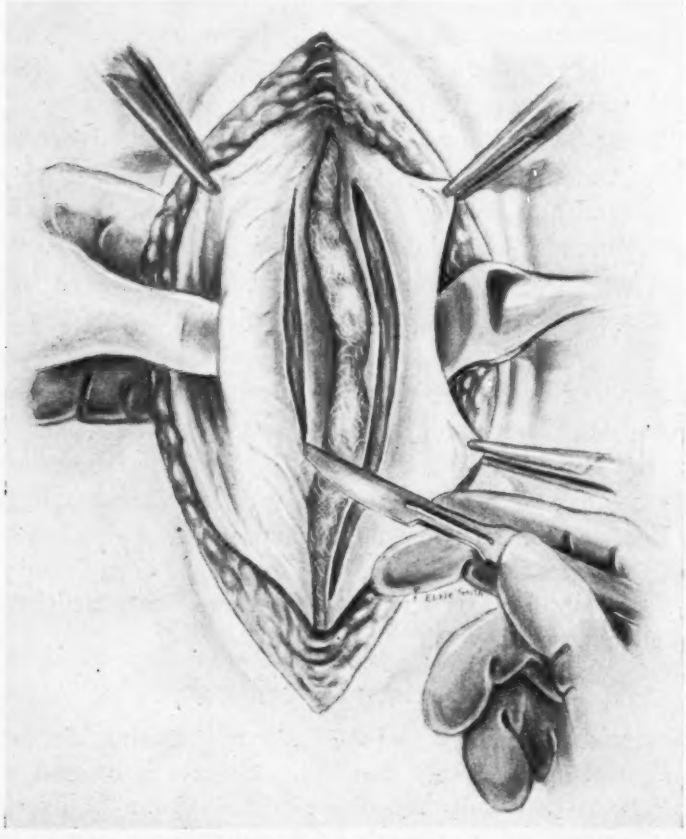


Fig. 2.—The sac has been incised and dissected free. The anterior sheath of the rectus has been exposed. The posterior sheath of the left rectus has been opened. The posterior sheath of the right rectus is now being incised.

ventral surface of the anterior sheath on the right side, carrying the dissection to a point just beyond the lateral border of the sheath. This procedure is repeated on the left side. The excess sac is cut away leaving a border of about three-fourths inch around the margin of the

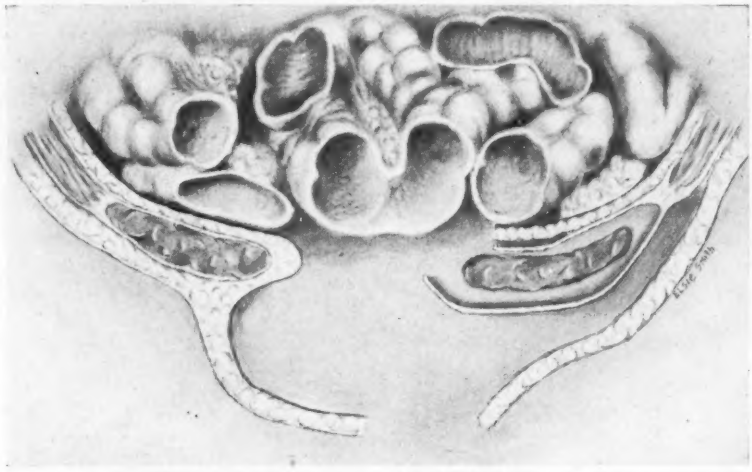


Fig. 3.—Cross section through hernia. The dissection has been completed on the left side. The right side has not been attacked.

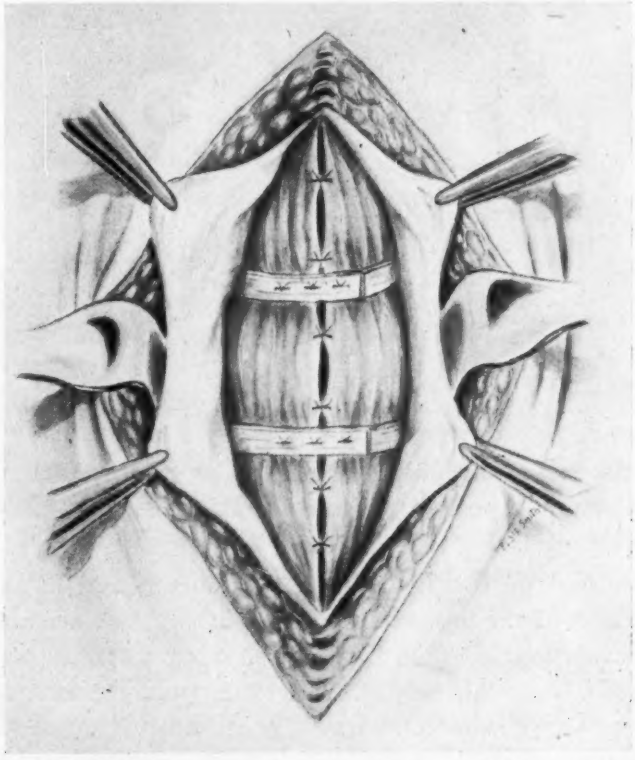


Fig. 4.—The posterior sheath has been closed. The rectus muscles have been dissected free and approximated in the midline with interrupted sutures and loops of beef fascia.

hernial opening. The anterior sheath of the rectus is not opened on either side as in most operations for ventral hernia. Rather, the posterior sheath is incised with the scalpel about one-half inch lateral to the hernial margin. The purpose of this posterior incision is to facilitate later closure as experience has taught that the posterior sheath can be approximated with much greater ease than the anterior sheath, and it is important in the dissection to save all the anterior sheath possible for later overlapping. This portion of the dissection can be more accurately accomplished if a grooved director is used.

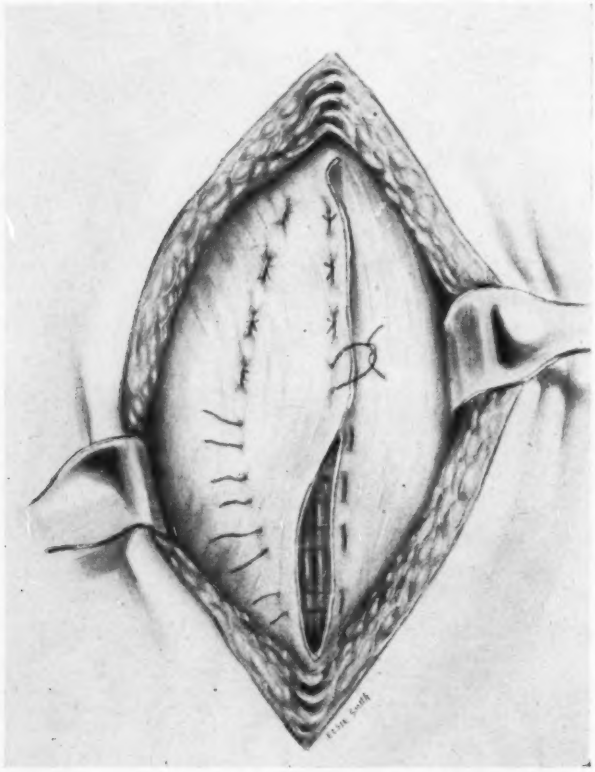


Fig. 5.—The anterior sheath of the rectus muscle is overlapped.

The rectus muscle on either side is gently dissected free of its bed, both anteriorly and posteriorly, care being taken to preserve its nerve and blood supplies.

Reconstruction of the Wall.—The peritoneum and as much of the attached fascia of the posterior sheath as is available are approximated in the midline, overlapping, if possible, using mattress or continuous sutures of No. 0 chromic catgut. After a thorough dissection this layer can usually be approximated without great difficulty.

The diastasis of the rectus muscles is then dealt with, the muscles being approximated at the midline and held in this position by two or

three loops of beef fascia which completely surround both muscles, but without tension (Fig. 4).

The ends of these fascia loops are overlapped and fixed by interrupted sutures of No. 0 chromic catgut. The margins of the rectus muscles are then approximated with interrupted sutures of No. 0 chromic catgut. The loops of beef fascia relieve the tension on these muscles which, of course, are extremely friable and prevent these stitches from cutting loose.

The anterior sheath of the rectus muscle is then overlapped, using mattress sutures of No. 1 chromic catgut (Fig. 5).

To summarize, we now have (Fig. 6) the peritoneum and the posterior sheath reapproximated. The two rectus muscles are approximated to each other in the midline by interrupted sutures of No. 0 chromic catgut reinforced to prevent lateral separation by loops of beef fascia. The anterior sheath of each rectus muscle is approximated to its fellow of the opposite side and overlapped in the midline.

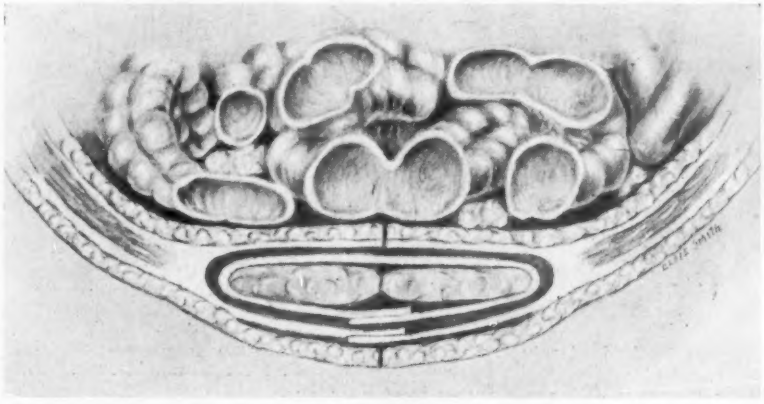
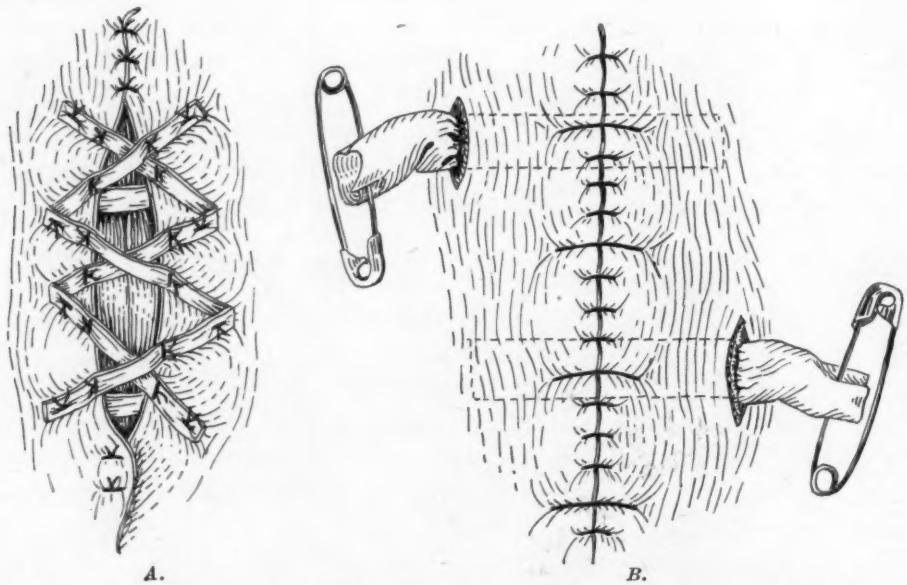


Fig. 6.—Cross section through reconstructed wall at the level of a beef fascia loop. Note posterior sheath approximated, rectus muscle approximated and surrounded by loop of beef fascia, and anterior fascia overlapped in the midline.

But, as frequently happens, regardless of how parsimonious the surgeon has been in his dissection, the fascia of the anterior sheath cannot be made to approximate completely in the midline (Fig. 7*A*). In this event, a portion of the approximated rectus muscle does not have an anterior fascial covering. Beef fascia is then sutured in a zigzag fashion back and forth in a basket arrangement to reinforce the fascial element of the abdominal wall at this point. The beef fascia is fixed to the anterior sheath by interrupted sutures of No. 0 chromic catgut.

Skin Closure.—(Fig. 7*B*.) The excess skin and fat are removed, and the wound is closed in the midline in the usual fashion. It must be borne in mind, however, that a large area has been undermined beneath the subcutaneous fat, and in spite of hemostasis some postopera-

tive oozing is to be expected. This will prove extremely troublesome in the healing of the wound unless adequate drainage is provided. Various methods of drainage have been tried by us, and it has been found that the most satisfactory results have followed the use of two transversely placed latex drains. One is introduced through a stab wound situated at the right of the superior angle of the wound and the other to the left of the inferior angle of the wound. The tubes are gradually withdrawn, a small amount each day, until the serous drainage has ceased. A snug abdominal binder is applied and worn during the hospital stay.



A. *B.*

Fig. 7.—*A.* Method of reinforcing edges of the anterior aponeurosis with strips of beef fascia. Fascia lata might be used instead. *B.* Skin wound closed. Two transverse latex drains introduced through stab wounds.

Postoperatively, an abdominal belt should be worn from six to eight weeks. Beyond this period very little is to be gained from its use and it is felt that active exercise to the muscles should be encouraged.

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SIMPLIFIED METHOD FOR FORMATION OF AN ARTIFICIAL VAGINA BY SPLIT SKIN GRAFT

Report of a Case

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CONGENITAL absence of the vagina is associated usually with a nonfunctional uterus. For this reason, symptoms of retained secretion, often observed in acquired vaginal atresia, are seldom seen in these cases. A person with complete absence of the vagina, who has only rudimentary internal organs, may be otherwise fully developed, with manifestations of secondary sex characteristics and normal external genitals. These women may have normal sexual instincts, and in many instances are quite attractive. To deny these unfortunate individuals the opportunity for the pursuit of normal activities because of the absence of an organ the construction of which would be a comparatively simple surgical procedure, is to subject them unnecessarily to an abnormal social life. Certainly, with the procedure now improved and simplified, and with its associated low mortality, there is every justification for surgical construction of a vagina in those cases of congenital absence which are otherwise normal.

It is commonly believed that congenital absence of the vagina is rare, but this anomaly is probably more common than general observation might indicate, because some women with this condition never present themselves for treatment and many others are probably unrecognized. Flynn and Duckett[1] estimated that fully 500 cases of surgical correction for this abnormality have been reported in the literature. Engstad,[2] who has operated on nine such patients, estimates from his own experience that this congenital deformity occurs once in every five thousand cases. Counseller[3] reports that the operation was performed sixteen times at the Mayo Clinic within a period of two years. Word[4] has seen three cases within fourteen months. Frank[5] reports that in twelve years he has seen more than thirty cases. In contrast, Newell,[6] who is affiliated with an institution which admits thousands of women, has had only four patients in twenty-five years. At Charity Hospital in New Orleans, over a thirty-four-year period, this condition was found only six times in 502,081 female admissions, 123,676 of which were obstetric and gynecologic patients.

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The variety of procedures for constructing an artificial vagina appearing in the literature is suggestive of the diversity of opinion which exists concerning the best operative technique. Roughly, this condition may be corrected surgically in one of three ways: by intestinal segment transplant, either utilizing the small intestine (Baldwin[7]) or the rectum (Schubert[8]); by pedicle transplant (Heppner[9]); or by free skin graft (Abbe[10]). Recently, Frank[5] advocated a means of forming an artificial vagina without operation. This he does by gradually forcing inward the mucous membrane in the introital region without an incision. He has treated six patients in this way with one failure. However, one must question whether these results were obtained in cases where there was not a complete absence of the vagina but where there was a small rudimentary canal.

According to Dannreuther,[11] Paunz,[12] and others, Dupuytren attempted to establish a vaginal canal as early as 1817, by making an opening between the bladder and rectum into which a tampon was inserted. Since then various attempts have been made to improve the technique of construction, although the fundamental principle on which the operation is based has not changed.

In 1898, Abbe[10] described a technique for construction of a vagina utilizing Thiersch grafts from the thigh applied over the surface of a rubber form, which in turn was inserted into the rectovesical space, which had been previously dissected out. The rubber form is removed in about ten days and contracture is prevented subsequently by plug bougies. This technique gave good results in one case. In a previous case Abbe had used the labia minora, but this was unsuccessful.

Baldwin[7] was among the first to suggest intestinal transplantation for the formation of a vagina. In 1904, he conceived the idea for this operation, but was unable to try it out until 1907. Through an abdominal incision, a segment of the ileum was severed and carried down into the dissected tract prepared for the formation of the new vagina. The vagina, thus constructed, seemed normal in every way. Haberlin[13] improved Baldwin's technique by implanting a loop of small intestine. This method of intestinal transplantation is undesirable, particularly because of the high mortality rate. Monod and Iselin[14] estimate a 21 per cent mortality following the operation. The intraperitoneal approach is probably largely responsible for this. Death in these cases has also been attributed to mesenteric thrombosis resulting from undue tension. Another disadvantage which the Baldwin technique incurs is the profuse mucous secretion following the procedure.

According to Schubert,[15] Gersuny, in 1897, was the first to utilize a membrane cut from the rectum to line the vaginal canal. He first lined the perineum and sphincter ani with a strip of mucous membrane cut from the anterior wall of the ampulla without interrupting its connection with the rectum near the plica vesicorectalis. This was

sutured on the posterior wall of the bladder as far as the orifice of the urethra. Gersuny operated on three patients in this manner with satisfactory results, although it was necessary to wear a conical dilator to prevent narrowing. This, of course, is an objectionable feature.

Sneguireff,[16] in 1904, divided the rectum in the sacral region, the lower end formed into a cul-de-sac, the upper end attached by suture to the skin, thus forming the artificial anus. The bridge between the anus and the vulva was divided and the wound closed so as to form a channel leading to the rectal segment. This method has been criticized because it forms a preternatural anus.

Schubert,[15] in 1914, and Popow,[17] in 1918, transplanted the lower rectum into the vulva, the upper rectal segment being utilized for reestablishing the continuity of the intestinal canal. The chief objection to this method is the comparatively high mortality rate (estimated at from 6 to 17 per cent). Monod and Iselin[14] give a mortality of 8 per cent with Schubert's technique. Death is often the result of peritonitis. Other disadvantages of the method are the high incidence of fecal fistulas and rectal incontinence developing after this operation.

Graves,[18] in 1917, revised the technique of flap transplant from the labia and thighs, which Heppner[9] did in 1872. A transverse incision is made below the urethra and a canal between the bladder and rectum is made by blunt dissection. Each labium minus is dissected downward so that the entire mucous surface is utilized and the pedicle is large enough for good circulation. Two skin flaps are then taken from the medial aspect of the thigh and the four flaps are sutured together over a glass form. The form is removed and the mass is invaginated into the newly formed canal, where it is held in place by catgut sutures. The canal is then packed with tampons. The most undesirable feature of this operation is the fact that if the flaps necrose, there is no more tissue for a second attempt. It has, however, the advantage of an extraperitoneal approach.

Because of the grave risk involved in the Baldwin and Schubert operations, Frank and Geist[19] devised a modification of the tube flap technique described by Gillies.[20] A tube pedicle skin flap is formed in the usual way. This is detached at its distal pedicle, split open, and wound around a hard rubber vaginal canal, which has been previously formed by blunt dissection. In 1932, they reported four cases in which this technique was followed, all of which resulted in successfully formed vaginas. Two outstanding disadvantages of this technique are the prolonged hospitalization (about ten weeks) and the defects over the inner aspect of the thigh at the site where the pedicle was formed. Among advocates of this method are Douglass,[21] Davis and Cron,[22] Grad,[23] and others.

In 1936, Monod and Iselin,[14] after bluntly dissecting a space to conform to that of a normal vaginal cavity through a transverse incision

between the urethra and rectum, applied Thiersch grafts over a hard stent, which in turn was inserted into the cavity. They report one case with good results. In 1937, Burger[24] proposed lining the artificial cavity with fresh fetal membranes.

In 1938, McIndoe[25] suggested to Counseller[3] an improvement in the skin graft technique to overcome contracture, which had been the reason for so many previous failures. A Thiersch graft removed from the thigh is wrapped around a mold and this, in turn, is introduced into the vaginal space which has been previously dissected out. The vaginal orifice is closed by approximating the labia minora over the mold, which is worn for a few weeks. Counseller[3] reported sixteen operations in two years at the Mayo Clinic with excellent results in all but one case. This operation has given better results than any of the previously described procedures.

In 1938, Wharton[26] offered a simplified operative technique for constructing a vagina based on the principle that the vaginal epithelium has remarkable powers of proliferation. The operation consists of two steps: dissection of a space between the bladder and rectum, and the insertion of a vaginal mold covered by a condom. This mold is allowed to remain in place three weeks. He performed the operation in two patients and also reported two other cases which were treated in the same manner by Burch and Hundley. The late results in two cases were entirely satisfactory; in the other two, only immediate results were known. However, he recommends this procedure not for cases of complete absence of the vagina, but for cases where a small rudimentary vagina is present.

In the light of the difficulty encountered in attempting to epithelize completely a newly formed vaginal cavity by means of a half thickness skin graft, it is not easy to understand the facility with which the entire cavity was epithelized solely by the proliferation of vaginal mucosa within the relatively short length of time stated. In our case approximately 85 per cent of the newly formed vaginal canal was lined by the half thickness skin graft applied at the first operation. A small fingerlike projection of granulation tissue resulting from failure of some of the graft to take, extended from the border of the vaginal mucosa at the orifice backward into the vaginal vault. There was another defect at the dome of the reconstructed vaginal canal. No attempt was made to regraft these areas for fifteen weeks, in the hope that these small defects would epithelize. However, this did not occur and proliferation of epithelium took place here more slowly than in less contaminated areas. It was noted also that the proliferation of epithelium over the fingerlike projection of granulation was no more rapid than that in the deepest portion. Because of these observations and because of the behavior of proliferated epithelium elsewhere in the body, I see no reason for expecting epithelization of a reconstructed vaginal cavity

by proliferation of epithelium from vaginal mucosa during a time interval which is much shorter than that for the epithelization of an equal surface defect elsewhere.

It can readily be seen that the ideal technique has not yet been devised. The procedure of choice should be simple in performance, should involve as little danger of complications as possible, and should give satisfactory and permanent results. Whereas we are cognizant of the necessity of modifying the procedure to suit the individual case, the basic principles involved in the different procedures are conducive to analysis and evaluation. Reconstruction of the vagina by intestinal segment transplant is objectionable not only because of the high mortality but also because of the possible development of serious complications, such as peritonitis, thrombosis of the vessels in the mesenteric segment, perforation of the bladder and rectum, and extensive fulminating infections. The procedure which utilizes the surface of the labia majora as an included segment of mucosa requires more extensive dissection than some of the simpler procedures. Similarly, those methods which employ single pedicle or tube pedicle skin transplant not only require lengthy operative procedures with danger of possible infection but also necessitate extended periods of hospitalization. Another disadvantage is the unsightly scars on the inner surface of the thighs which must be subsequently repaired. The newly formed vaginal canal will probably be less satisfactory with the use of full thickness skin than with half thickness skin graft. The simpler methods which establish a vaginal canal by blunt dissection and effect proliferation of vaginal epithelization have already been discussed.

Therefore, the procedure which offers the best results with the least amount of operative maneuvers and minimal risk is the one which establishes the vaginal canal and affords a lining for it by the application of a half thickness skin graft. Among the factors which determine the successful take of skin grafts are an adequate blood supply, freedom from infection, and adequate and constant pressure. The first two factors are supposedly constant in many of these procedures. The last factor is greatly enhanced by the use of a vulcanite form of proper measurements, so carried as to distribute adequate and uniform pressure. We propose a modification of the skin graft transplant technique which is simple, safe, and satisfactory.

A transverse incision is made between the urethra and the anus at the point where the normal opening of the hymen is usually found in the virgin. Through this incision, with the index finger, a vaginal canal is dissected out bluntly between the rectum and the bladder extending upward to a normal depth. Extreme care is taken not to penetrate the peritoneal cavity. A half thickness skin graft is then removed from the inner surface of the thigh, provided this region is sufficiently hairless; otherwise, it is taken from the inner aspect of the arm. The skin

graft is then wrapped around a smooth vulcanite form, which has been made prior to operation, and the entire mass is inserted into the newly formed vaginal cavity. The piece of skin should be large enough to completely cover the form. The measurements of the cast (10 to 12 cm. long and 12 cm. in diameter) conform to those of a normal vaginal canal. It is cylindrical in shape and rounded at both ends, with a slight concavity on one side in the center and a proportionate convexity of the other. To the distal end (the portion which protrudes through the vaginal orifice) is attached a slight knob, not unlike the knoblike portion of a dumbbell. Through this knob are made two holes for the reception of rubber tubes, which are used to hold the vulcanite form firmly in place after it is inserted into the vaginal canal. These tubes exert the proper pressure for maintaining the form under suitable tension in a desirable fixed position. The two ends of the rubber tube are

Fig. 1.—Photograph showing the normal anatomic appearance of the labia minora and majora as well as that of the clitoris. Note that there is no suggestion of a hymen or rudimentary vaginal canal. A firm diaphragm covered with mucosa is present. There is no dimpling.

attached to an abdominal belt anteriorly and posteriorly. The tube passes anteriorly downward through the opening in the mold between the thighs and then follows the gluteal fold to pass upward posteriorly. When the tubes are in place, they act as a perfect suspensory sling. These not only support the vulcanite form but hold it constantly in place by conveying elastic pressure through its contact with the form. The form, when worn, is nonirritating and is not uncomfortable to the patient. Because the vulcanite form has been made to conform to the size of the newly made vaginal canal, and because the pressure is properly applied by the rubber tubes acting as a sling support, the graft is held snugly in place in the vaginal canal and is kept in intimate contact

with the denuded surface of the canal as well as being maintained in a fixed immovable position. The patient is kept in bed in a prone position for approximately ten days, after which the form may be withdrawn, so that the newly lined vaginal canal may be cleansed and irrigated. The form not only maintains the desired size of the vagina, but it acts in intimate contact with the graft to support and constantly massage it, this latter action rendering the graft pliable and soft.

At the end of approximately two weeks, the graft has healed sufficiently to permit the patient to sit up in a chair with the form still in its fixed position. No discomfort whatever is experienced from the constant wearing of the vaginal cast. When strength permits, the patient

Fig. 2.—Photograph showing the vaginal form inserted in the newly formed vaginal canal. Note the appearance of the rubber tubes where they pass through the openings in the knoblike portion of the distal end of the vaginal form.

resumes normal activity with the dilator constantly in place. Because of the ease with which the vulcanite form may be removed, daily douches may be taken and frequent inspection with cleansing of the canal may be made. The above routine insures normal expansion of the canal so that the desired pressure is constantly applied to the surface of the graft. Because of the action of the vulcanite form against the graft, the entire lining surface of the canal is constantly massaged, thereby rendering it pliable and soft. The form should be worn for about six months in order to avoid the possibility of contracture, which inevitably results if the surface is not constantly massaged for approximately that length of time.

The following case illustrates the technique described.

REPORT OF CASE

This patient (Case No. M1535), a normal, white girl, aged 30 years, was admitted to a local hospital, July 1, 1939, with a diagnosis of congenital absence of the vagina.

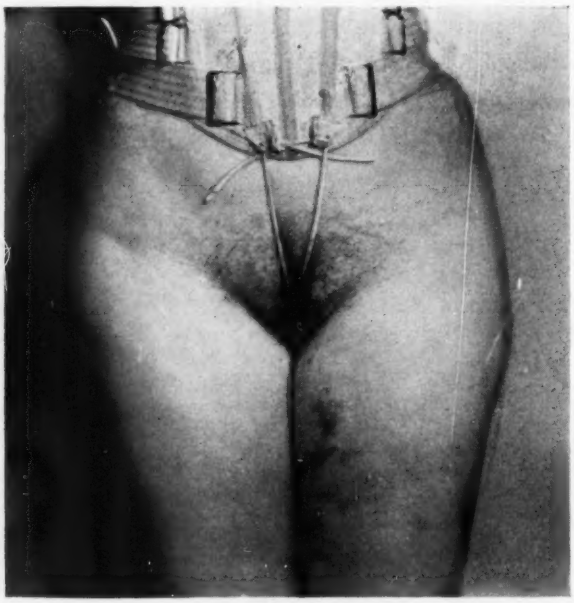

Fig. 3.—Photograph showing the attachment of the suspensory tubes to the lower medial border of the abdominal belt, anterior view.

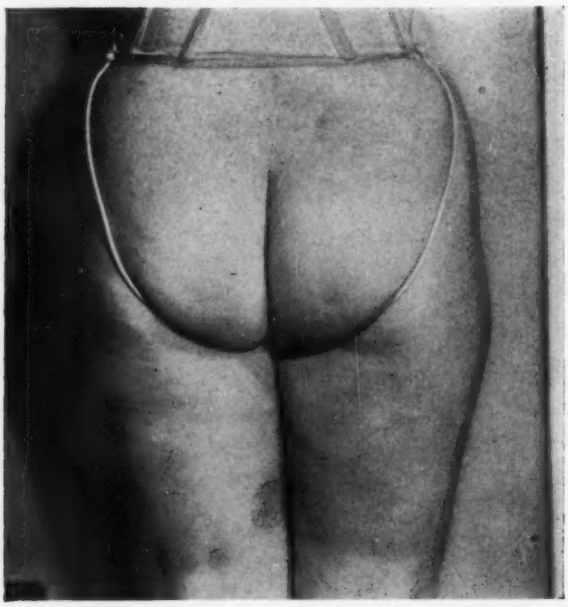

Fig. 4.—Photograph showing the suspensory rubber tubes, posteriorly, as they course upward to follow the gluteal folds. From Figs. 3 and 4, one can see the support which is afforded the vaginal form by means of the suspensory tubing. An adequate amount of upward pressure is conveyed to the vaginal form thus assuring intimate contact of the form with the graft.

The patient engaged in normal activities and was not aware of her abnormality until it was discovered when an examination of the perineum was made several years before. She had consulted surgeons at three clinics. At two of these the operation using the intestinal segment was advocated and at the other reconstruction by the creation of a vaginal cavity by blunt dissection and the insertion of a bougie was recommended. Because of the variation in the suggested technique and the prognosis, decision to have the operation performed was deferred for several years.

Physical examination revealed a healthy, attractive, white girl, 30 years old. Both breasts were normally developed. There were no tumors or abnormal growths. Female sex characteristics predominated. Examination of the vulva showed normal female distribution of pubic hair and the presence of the labia majora and minora. A surface of mucosa appearing as a flat normal indurated area lay between the labia minora. Complete absence of the vagina was indicated by the absence of indentation. Rectal examination disclosed a small tumorlike growth, which was probably a rudimentary uterus, and a resistance on palpation, which was apparently the left ovary. All other physical findings were normal. (See Fig. 1.)

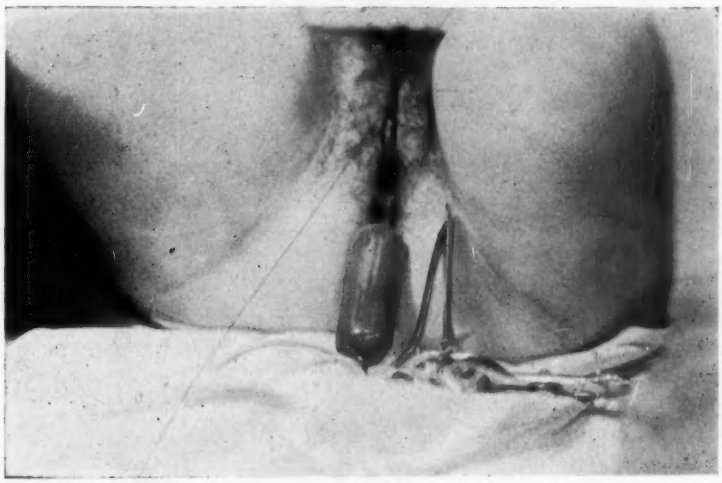

Fig. 5.—Photograph showing the vaginal form compared with a large vaginal speculum.

On July 1, 1939, under cyclopropane anesthesia, after thorough cleansing of the vulva with normal saline solution and soap, an incision was made between the labia minora from the urethra downward to a point where the fourchette is normally found. An opening was made upward and backward in the region where the vagina is normally found, and by blunt dissection this was carried back 10 to 12 cm. To avoid perforating the rectum one finger of the assistant was placed in the rectum and a retention catheter inserted through the urethra into the bladder. Following this a form of the newly constructed vaginal vault was made from a plastic dental compound and over this was wrapped a split skin graft which had been removed from the medial surface of the left thigh. This was introduced into the newly formed vault where it was held fixed by means of through-and-through tension sutures inserted in the tissues at the orifice. A sterile pad was then applied and the legs held strapped for about twelve hours. Following the operation catheterization of the patient yielded a considerable quantity of urine, indicating that the bladder had not been perforated. Vaginal examination at operation revealed the absence of a uterus, but the presence of the adnexa.

The patient's postoperative course was uneventful, the temperature never exceeding 101° F. and becoming normal within a few days. The vulva was irrigated regularly. An 85 per cent take of the graft was obtained. The patient progressed nicely and was discharged, July 23.

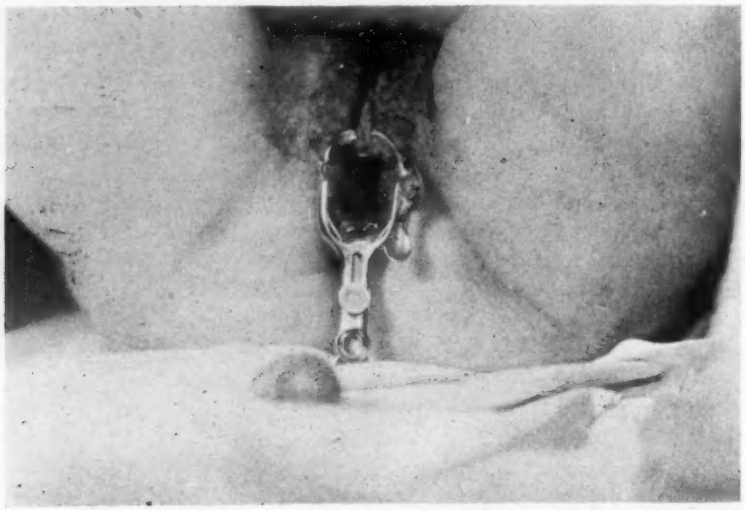

Fig. 6.—Photograph showing large vaginal speculum inserted into the newly constructed vaginal canal. Note the size of the canal after the speculum has been completely opened.

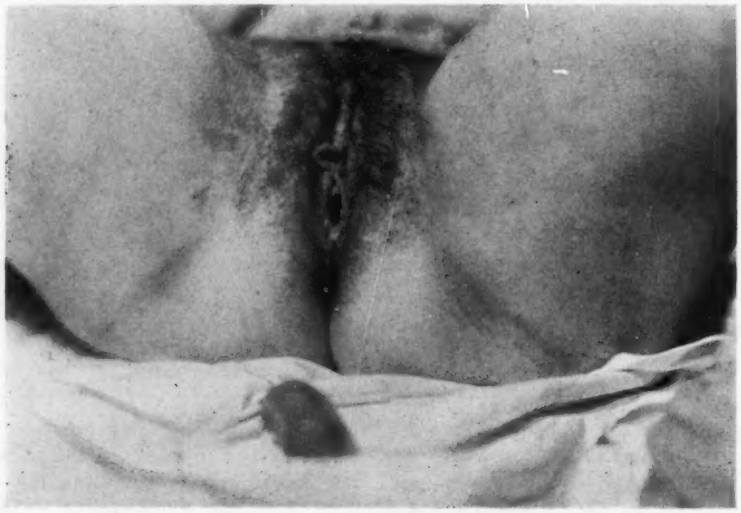

Fig. 7.—Photograph showing the normal appearance of the perineum following the construction of the vagina.

Due to the difficulty of retaining the form which had previously been made from a plastic dental compound, a vulcanite form was made to conform to the size of the newly made vaginal canal. A complete description of this form has already been given which enumerates the many advantages of this type of dilator over those which are commonly used. The patient was able to wear this form with comfort and because of the support afforded by means of the rubber tubes, a satisfactory

pressure was always applied. These tubes permit the form to be removed with ease and as a result of this frequent cleansing is permitted. The vulcanite mold is worn constantly during the first six months following operation. (See Figs. 2 to 5.)

Because of a slight area of granulation tissue at the orifice and another small area at the dome of the newly formed vaginal canal, on November 13, a second graft was removed from the left thigh and placed on the stent and the stent reinserted. A third graft was applied to a slight defect at the dome of the canal. Results following this procedure were satisfactory.

The patient was kept under constant observation for a period of six months, during which time she wore the vulcanite form continuously, changing it daily to permit cleansing and douches. At this time no tendency toward contracture was seen. The canal remained approximately the same size. The character of the skin graft which had been applied to establish a lining for the constructed canal showed definite change. In appearance the skin resembled mucous membrane. No microscopic section of this tissue was made since it has been shown by previous authors that skin grafts, when applied to defects in mucous membrane lined cavities, uniformly manifest a change which makes them not unlike mucous membrane in appearance. In some instances the microscopic picture shows a change which is similar to that seen in microscopic sections of mucous membrane.

Frequent examinations were made on this patient for two years and it was found that the character of the skin graft remained essentially the same as that described above and it showed no tendency to become unstable. No tendency to contracture occurred during this time. See Figs. 6 and 7.

SUMMARY

1. There is every justification for the construction of an artificial vagina in those cases of congenital absence which are otherwise normal.

2. Congenital absence of the vagina is not as rare as formerly assumed.

3. The various operative techniques for correction of this defect are reviewed briefly.

4. A modification of the skin graft transplant technique, which is simple, safe, and satisfactory, is proposed.

5. A case of congenital absence of the vagina in which this method was employed with excellent results is reported.

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Recent Advances in Surgery

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CRUSH AND COMPRESSION INJURIES WITH UREMIC DEATH

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DURING the past year the British medical literature has contained reports of cases which, at first, were thought to be unique observations of this war. These were cases of crush and compression injuries accompanied by renal failure which for a time were considered to present a new syndrome. It is incredible, however, that this syndrome has not been associated with injuries sustained in civil practice (mine, automobile, and other types of accidents resulting in crush and compression injuries). It must be that the effects of such injuries were not observed carefully or not recorded, inasmuch as very little has appeared on the subject in the English literature. The studies of Bywaters and others[5-7] have revealed references in German periodicals to cases occurring in the last great war.

During the Messinan earthquake, in Italy, a considerable number of persons were crushed, compressed, or buried. A German expedition arrived at the scene fourteen days after the catastrophe. Colmers,[8] in 1909, described among eighty-three casualties from this earthquake nineteen victims suffering from acute fatal pressure necrosis, and in only one was there a history of oliguria with blood urine.

In 1916, Frankenthal[14] described three soldiers who had been buried and who showed edema, bloody urine, and post-mortem ischemic muscle necrosis. However, no importance was placed on the findings in the urine. Other pathologists described similar material, some in journals now inaccessible. Among these Hackradt,[21] in 1917, from Borst's laboratory described tissue from a patient who had been buried for nine hours, and in whom occurred edema of the legs and vesicles and bloody urine containing albumin and casts. The patient died on the fifth day. Necropsy showed muscle necrosis with associated tubular degeneration in the kidneys. Hackradt focused attention on the sharp distinction between acute hemorrhagic glomerulonephritis (''war nephritis'') and the specific tubular degeneration in the kidneys associated with crush or compression injuries of the extremities and other parts of the body. He cites the autopsy findings of one case reported by Borst[2, 3] and two cases by Bredauer.[4] On the basis of autopsy findings, Hackradt points

out that such traumas, especially when they are multiple and involve the central nervous system, chest, abdomen, or pelvis, cause death as a result of widespread fatal embolism.

In microscopic studies of autopsy material the German authors found no disturbance in the circulation of the kidneys as evidenced by vascular engorgement. Brown corny cylinders were found in the tubules of the medullary substance and were considered to be the products of intense methemoglobin infarction in the pyramids. The marked reduction in the quantity of excreted urine was thought to be caused by blockage of the canaliculi with methemoglobin cylinders. Bredauer[4] attributes the presence of free hemoglobin in the blood and the excretion of hemoglobin in the urine to the disintegration of muscle substance at the point of muscle necrosis, the formation of catabolic products there, and the absorption and circulation of the myohemoglobin and foreign proteins in the blood stream.

Minami,[37] in 1923, summarized the literature and investigated the material from three other cases from Pick's laboratory, cases which had been described by Lewin,[25] in 1919. Minami's description tallies exactly with the findings of Bywaters and Beall,[5, 6] in 1941, in two victims buried by grenade explosion. On the second day following the event, these patients showed painful swelling of the thigh; on the fourth day, scanty bloody urine and tenderness in the kidney regions appeared; on the fifth day, only 100 c.c. of urine, less pigmented, was found; and on the sixth day, when death occurred, the urine was still scanty but not yellow, and contained red and hyaline casts. Necropsy showed grey muscle necrosis at the point of injury. The kidneys showed normal glomeruli, degeneration of the convoluted tubules, and pigmented masses and ribbons of debris in the collecting tubules and in Henle's loops. Minami pointed to the analogy between this condition and paralytic myohemoglobinuria of horses. He intended, were it possible for him to get fresh material, to identify the pigment spectroscopically. By the end of the last war the syndrome was well recognized by the Germans and was included in their textbooks of surgery. In Great Britain, however, it appears to have been unrecognized and undescribed prior to the present war. All such traumatic cases terminating in death should be investigated systematically, not perfunctorily.

In attempting to explain the peculiar muscle necrosis in compression injuries some interesting hypotheses have been advanced. Minami postulates that the muscle necrosis appears to result from indirect injury to the vessel walls without thrombus formation. As a result, a patchy, irregular distribution of the areas of necrosis occurs, not only in the lower extremities but in the muscles of the back, chest, abdomen, and buttocks, as well. Orth[38] explains the necrosis as a consequence of the indirect traumatic pressure. Wieting[46] believes that the necrosis is caused by a parenchymatous degeneration of the muscle elements. Groll[19] thinks that colloid-chemical, osmotic, acid penetration of the

accumulated products of metabolism into the injured muscles is the responsible agent. Frankenthal,[14] Borst,[2, 3] and Schmincke[44] are of the opinion that the muscle necrosis is of local anemic origin based on the vasomotor paralysis of the vasomotor nerves, and that the bleeding into the tissues at the point of injury is diapedetic in origin.

Acute renal insufficiency is a common clinical finding in cases with crush or compression injuries, as has been recorded by Bywaters and Beall (1941);[6] Beall, Bywaters, Belsey, and Miles[1] in 1941; and Mayon-White and Solandt,[30] also in 1941. The acute renal insufficiency is always accompanied by acute changes in the kidneys of a specific and unusual nature. The most definite changes found in the kidneys from the crush syndrome comprise the following: (a) light-brown casts resembling hemoglobin in secondary convoluted tubules; (b) proliferation of cells in some of these tubules; (c) the more intense focal lesions rupture the tubules and allow protrusions of albuminous material into venules; (d) large orange-brown casts are found in the medulla in many collecting tubules. Their presence is associated with damage and regeneration of the epithelium, and the casts are quite numerous. The essential pathologic lesion is restricted, then, to the ascending limbs of Henle's loops and the second convoluted tubules. The most severe of the tubular lesions implicates the walls of the small venules into which many of them have perforated.

It is difficult to find in the diseases of man any analogy for a condition of selective damage to the lower segments of the nephron, but evidence drawn from experimental lesions in animals may assist in elucidating the pathogenesis of a lesion of this kind. Dunn and Polson,[13] in 1926, demonstrated that by introducing into the circulation of the rabbit large doses of uric acid in solution in the form of lithium monourate, selective damage could be produced in the ascending limbs of Henle, in the secondary convoluted tubules, and to some extent in the collecting tubules, while the first convoluted tubules remain normal in appearance. Necrosis of the epithelial linings is produced within an hour or so after injection, and the regenerative changes are in full swing by the fourth to the eighth day. In the regenerative phase the lesions show a distinct similarity to those seen in the kidneys of cases with crush or compression injuries. In these animals, just as in uranium or oxalate nephritis where the first convoluted tubules are selectively damaged, an anuria or oliguria is present with a rise in blood urea. These findings have been attributed by Dunn and Polson to nonselective reabsorption of filtrate through the damaged wall of the tubules into the venules thus escaping excretion. More recently, McFarlane (1941)[27] has shown that in the experimental nephritis of rats, which was described by MacKay and Oliver,[28] in 1935, and by Duguid,[10] in 1936, the lesion is again restricted to the lower nephron segments, and he agrees with Dunn and Polson in attributing the special localization at this level to change of reaction in the filtrate.

A feature which appears to be unique among the tubular changes in the reported cases is the localization of the most intense damage at points where the lower nephron segments lie in close proximity to veins. In uric-acid nephritis there seems to be little doubt that the lesion is determined by precipitation of the agent from a soluble form in which it is relatively harmless to a solid form which exerts the destructive effect. It seems possible that in the cases reported, also, a specific toxic substance is produced in the presence of acid, and that the proximity of venous blood has an influence in permitting a greater swing toward an acid reaction in this area than is reached at other parts of the nephron.

Nothing certain is known, as yet, of the nature or source of any toxic agent which may be responsible for the renal damage in cases of crush syndrome. The available facts, however, appear to justify the primary view to which most observers have adhered—that after release of the affected tissue from a compressed or more or less ischemic state, something toxic is absorbed from it into the circulation as the vessels reopen, and that the toxic agent affects the kidney in the course of excretion. The number and kinds of possible toxic agents which could so arise are conjectural and the suggestion that the cast material, which apparently contains myohemoglobin (Bywaters and Delory,[7] 1941), is toxic is probably true. Uric and phosphoric acids have been suggested as possible toxic agents to the kidneys, since both of these molecules have a well-recognized origin in muscular tissue in the form of inosinic acid.

The part, if any, which may be played in the initiation of this syndrome by the lowered blood pressure in shock has yet to be determined. Bywaters and Beall,[6] while recognizing that a fall of systemic pressure and dehydration may contribute toward the development of oliguria, could not attribute primary significance to these factors in their cases or those of others. There also appears to be general unanimity that the degree and kind of renal functional disturbance which develop after the crushing of limbs are quite distinct from any of the sequelae of ordinary surgical shock. The significance of selective involvement of the kidneys in the crush syndrome can hardly be overlooked.

In the records of fatal cases of crush injuries the brown casts in the renal tubules have been a conspicuous feature, and similar casts have been described in the urine of patients who recovered (Longland and Murray, 1941[26]). The question has been raised as to whether blockage of tubules by these casts could be the cause of the renal disability, but histologic evidence on this point is inconclusive. The persistence of casts in the urine could easily depend on the diminution of the filtrate, whether this resulted from an impaired circulation, dehydration, or some other cause. The most important criticism of the blockage theory, however, has been made by Bywaters and Beall,[6] who point out that if tubular obstruction alone were concerned, such urine as was secreted should theoretically be of normal composition, coming from unobstructed tubules, but in their experience the urine was little more than a glo-

merular filtrate with a urea concentrating factor of 1.6 to 3.8 in their Cases 1 and 2. This is confirmed by figures taken from Henderson's[22] (1941) chart of a recovered case with a concentrating factor about 3.3 on the tenth day when the urinary output was about 700 c.c. In Cases 1 and 2, reported by Professor J. Shaw Dunn and others,[11] single observations on the eighth day gave concentration factors of only 2.7 and 3.3, respectively. Such extreme degrees of ablation of one of the highest functions of the kidney are of considerable moment and it is no doubt significant that among acute renal conditons the greatest reduction of concentrating power is known to occur when there is severe damage to tubular epithelium. This may be observed in mercurial nephritis in man and animals.

The clinical picture of fatal renal failure after crushing or compressing of limbs or other parts of the body, or after burns, is best presented by describing a typical case history: The patient has been buried for several hours with pressure on a limb or limbs. On admission to the hospital his condition may be good, the pulse and the blood pressure normal. There is swelling of the limb, some local anesthesia, and whealing. The edema spreads along the limb or limbs, movements of the limbs become limited or are absent, and various sensory disturbances may be present, such as anesthesia, hyperesthesia, or paresthesia.

In the crushed limb the changes observed include necrosis of the muscle of patchy distribution, hemorrhage into the muscle which may not be constant, edema of the limb, and an increased tension of the tissues within the fascial compartments of the limb. Intense arterial spasm appeared to be a common cause of the ischemia. Clinically, peripheral arterial pulsation was sometimes absent when the case was first seen. Loss of nerve function was recorded in the majority of the cases.

The hemoglobin is raised; a few hours later the blood pressure falls and shock ensues. This may be successfully combated by transfusions of serum, plasma, or blood. The urine, however, diminishes and contains albumin and many dark brown and black granular casts. The patient is alternately drowsy and anxious. Thirst and excessive vomiting are present commonly. The blood urea and potassium become progressively higher, the sodium chloride and the CO_2 combining power of the blood are progressively diminished, and death occurs in about a week.

Necropsy reveals necrosis of muscle and, in the renal tubules, degenerative changes and casts containing brown pigment. The casts, on microscopic examination, appear to be composed not of red cells but of desquamated epithelial cells, the pigment probably being due to hemoglobin excreted into the lumen from the blood stream. The gross and microscopic appearance of the kidney resembles the kidney of mismatched transfusion. There was, however, no clinical evidence of a transfusion reaction, and samples of plasma taken seventeen and forty hours after transfusions showed no increased color. Furthermore, fatal

cases with identical lesions in the kidneys are being reported, in which blood transfusion has not been given. Bywaters and Beall[6] point out that the syndrome did not occur in any of twenty-five shocked and injured patients without severe muscle crush, treated in their hospital by blood or serum transfusion. The only common factor in all of the reported cases is muscle necrosis.

The prognosis depends upon many factors. The duration of the crush did not seem to bear much relation to survival. Those crushed for a long time might recover. Another factor influencing prognosis was the extent of the injury. Particular care should be exercised in a case of muscle damage by crush or compression, for at the beginning the serious cases can not be distinguished from the comparatively minor ones. A further factor was the rise in the blood urea. In the severe cases in which the patients recovered there was a fall in the blood urea at about the same time as death occurred in the fatal cases. The urea could rise to very high levels, and the patient might recover without any treatment whatever. Recovery occurred in about one-third of the patients admitted. In about two-thirds of the cases, death took place from renal failure, generally at about the fifth to the eighth day.

Treatment is directed toward dilution of the toxins and promotion of diuresis, while adrenal cortex extract is given to check the rise in blood potassium which results from the increased permeability of injured muscle, at the site of injury, and the more rapid escape of intercellular ions. Primary amputation of the affected limb has been tried in a few cases but has failed to save life, while in the others when recovery occurred, it did so without amputation. Decompression of the limb by multiple incisions into the limb was tried in a few cases without results. Patey and Robertson[39] thought the syndrome might be due not to liberated autolytic bodies from damaged muscles, but to a loss of substances from the circulation into the affected limb on the release of compression. This would readily explain the shock and possibly the renal failure as a secondary manifestation of shock. If this is true, large intravenous injections of plasma or serum would tend to increase the loss of blood constituents into the damaged limb in the same manner as in a burn, with subsequent increase in edema. The logical procedure seemed to be to prevent this leakage of fluid from the circulation. Patey and Robertson treated two cases by applying intermittent positive pressure up to 60 mm. of mercury to the edematous areas, using an especially large blood pressure cuff. In both cases there was a greatly increased output of urine immediately, softening and later disappearance of the edema, a fall of blood urea to normal, and recovery. On the basis of this finding, it would be logical to bandage the limb firmly by wide bandages immediately at the time the debris is removed from the limb. The limb should be elevated and splinted as soon as possible. For the associated peripheral spasm probably heat and intravenous atropine and papaverine are indicated.

It should be impressed on rescue workers that the real danger to people pinned down by wreckage is kidney failure, and therefore it is of paramount importance to ensure copious diuresis, particularly at the time they are about to be released from the pressure. The advantages of a good urinary flow would be a dilution of chemical substances in the kidney tubules and perhaps prevention of precipitation of myohemoglobin casts. As pointed out by Maitland,[29] in 1941, isotonic sodium sulfate solution has been advocated as a potent diuretic from time to time. In view of the original oliguria present, he suggests that isotonic sodium sulfate solution be given by the drip method, intravenously, up to several pints a day.

No severe additional damage or difficulty would accrue by the immediate application of a tourniquet above the compression. This should be maintained until good diuresis has started, then the tourniquet should be released gradually.

These patients have a low alkaline reserve and alkalinization of the patient should be instituted as early as possible. The administration of alkalis in quantities adequate to render the urine alkaline is a rational procedure. Sodium bicarbonate and sodium citrate should be given by mouth from an early stage in quantities of 200 or 300 gr. a day. The use of a soduim salt must be emphasized because of the high blood potassium present which might conceivably be one of the biochemical factors leading to death. Improvement of urinary output occurs after extensive alkaline therapy. Good diuresis, of course, cannot be maintained in the presence of circulatory collapse. Hence, shock should be treated by transfusion, the transfusion fluid being serum or plasma.

CONCLUSIONS

1. The admission of a large number of casualties to a hospital may result in the early crush syndrome being overlooked unless examination is systematic. The patient may look well in the early stages and be passed over temporarily.

2. To date, the present war has produced no new surgical problems. However, one outstanding observation has been made—the appearance of grave and often fatal kidney damage after prolonged compression of the limbs.

3. Most authors agree that the essential pathologic lesion is muscle necrosis at the site of the injury; that the breakdown of autolytic products from dead or dying muscle is the main factor which damages the kidneys. Two main effects are produced in the kidneys: Some chemical substance is liberated which poisons the tubule selectively, and there is present, also, a tubular blockage factor, quite possibly resulting from the precipitation of myohemoglobin casts.

4. It is generally agreed that the crush syndrome is a definite entity and though resembling mismatched transfusion and the effects of prolonged shock, it is differentiated by its distinct clinical, biochemical, and pathologic features.

5. Primary amputation in the early stages has been tried in a few cases and has resulted fatally. Decompression of the limb by multiple incisions has also been tried in a few cases without result. Therapeutic attempts are directed toward the treatment of shock, dilution of the toxins and promotion of diuresis, restoration of a depleted alkaline reserve, compression by intermittent positive pressure or by firm bandaging of the injured limb, immobilization of the injured part and intravenous atropine and papaverine for release of the associated arterial spasm.

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Book Reviews

A Manual of the Treatment of Fractures. By John A. Caldwell, M.D., Professor of Clinical Surgery, College of Medicine, University of Cincinnati; Director of the Fracture Service, Cincinnati General Hospital. Pp. 150, with 76 illustrations. Springfield, Ill., 1941, Charles C Thomas, Publisher. $3.50.

This small concise manual of the treatment of fractures is probably the best of the several such books that have been published in the past few years. It fills adequately the need of medical students, house officers, and the general practitioner for a small but up-to-date authoritative textbook for rapid reference. It adheres to the recognized orthopedic principles and makes clear what leading contemporary authorities consider good practice in the treatment of fractures.

The illustrations are ample, to the point and diagrammatic. Unfortunately, there are no roentgenographic reproductions.

At the end of each chapter there is included a short list of references to the pertinent literature which should be of value to the student wishing to review the original sources. The book can be recommended as a welcome addition to our teaching armamentarium.

Synopsis of Genitourinary Diseases. By Dodson, Austin I., M.D., F.A.C.S. Ed. 3. Pp. 302. St. Louis, 1941, The C. V. Mosby Co. $3.50.

This small, compact text is, as its name implies, a condensed handbook on urology. For lack of space, many theoretical considerations and alternatives of treatment are necessarily omitted, but it must be emphasized that nothing of any real practical value is left out. Also, surgical technique and postoperative care of surgical cases do not come within the scope of such work, and properly so, since it is designed purely as a handbook or guide in the management of office urologic practice.

The present edition is the third, and appeared last year, indicating the Dr. Dodson has revised and brought the book completely up to date, as regards recent advances in treatment.

All in all, this little text can unhesitatingly be recommended to all students and general practitioners of medicine as perhaps the finest work of its kind, in bringing to them in easily accessible manner, the innumerable bits of information necessary to the office practice of urology.

Medical Progress Annual 1940. A series of fifty-two reports published during 1940 in the *New England Journal of Medicine* under the managerial editorship of Robert N. Nye. Pp. 625, with 8 illustrations. Springfield, Ill., 1941, Charles C Thomas, Publisher.

Their monograph presents an excellent group of papers, requested from the *New England Journal of Medicine*, written by foremost men in their respective fields. The book embraces most of the specialties of medicine. A number of papers of great interest to the surgeon are included: A. W. Allen writes on "Progress in Abdominal Surgery"; W. E. Ladon on "Children's Surgery"; C. M. Jones on "Gastroenterology"; J. D. Stewart on "The Nutritional Requirements in Obstructive Jaundice"; R. H. Smithwick on "Surgery of the Sympathetic Nervous System"; and E. D. Churchill on "Progress in Thoracic Surgery."

This list of authors, as well as many equally well known in other fields, gives assurance of the authoritative character of the reviews. Obviously, too, as the title implies, key indices to pertinent current literature are included. This monograph complements, in a useful manner, the year books published in the various specialties. This reviewer believes this effort of the authors, editor, and publisher to be a worthwhile endeavor which should be continued from year to year.

Psychosurgery. By Walter Freeman and James W. Watts. Pp. 337. Springfield, Ill., 1942, Charles C Thomas, Publisher.

This challenging monograph is devoted to the operative treatment of patients with intractable psychoses. Part I gives an interesting historical account of the early efforts of surgeons to deal with mental disorders, from the primitive trephining of the Incas down to the random cortical resections of Burckhardt in Switzerland, fifty years ago. It remained for Puussepp in Estonia and Egas Moniz in Portugal to develop a more rational plan of interrupting the frontal association fibers. Part II takes up the anatomic-physiology of the frontal lobes and their connections, through the frontal association pathways, with the medial dorsal nucleus of the thalamus and other portions of the brain. The function of the frontal association areas has been arrived at from many angles and the authors' description of psychologic observations made after experiments on lower animals, frontal lobe softening, atrophy, wounds, and the effects of lobectomy in man is a particularly valuable review of this field.

In Part III Freeman and Watts describe the technical improvements they have made in the Moniz method of frontal lobotomy. With the original "leucotome," transection of the entire frontal radiations was uncertain and it soon became obvious that there are definite hazards in transecting the fiber tracts from above. The authors' present technique of making bilateral trephine openings (through the coronal suture and 6 cm. above the zygome) and cutting across a wide pie-shaped section of white matter with a straight, blunt instrument permits a surer and safer division of the association fibers. The operation is carried out under regional anesthesia, which permits continued observation of the psychologic reactions of the patient. These are described in interesting detail and special emphasis is laid on the fact that the surgical transections must be made to a sufficient extent to produce drowsiness on the operating table. Freeman and Watts' method, however, is not free from cerain risks, as three patients have died as the result of postoperative clots and a similar number have developed epilepsy.

The authors begin their work with the obvious, although unfortunately not generally accepted, assumption that psychiatric disease is brain disease. To this is added a second assumption, namely, that brain surgery is therefore a reasonable therapeutic approach. The interesting points in the book from the psychologic point of view are: (1) the changes during operation, (2) changes of thinking and behavior caused by the operative lesion, (3) changes in the patient's illness following operation, that is, the therapeutic results.

It is striking that a change in the patient's state of awareness of his surroundings and ability to report on it takes place only after the operative lesion has been made in upper and lower quadrants bilaterally. The end point for the "complete" operation is a psychologic, not an anatomic one, that is, confusion or impaired ability to respond on the part of the patient.

As a result of operation there are certain noteworthy common changes in the patients: (1) loss of ambition and drive, (2) change in emotional attitude, and (3) deterioration in social habits. The authors describe "lack of initiative," "procrastination," "slowness, laziness, dullness, fatigability, euphoria, apathy, factitiousness, irritability," "tactlessness, poor judgment, sarcasm," and "impulsive acts,"

as examples of change or loss. In other words, new symptoms are produced and presumably mark an improvement over the symptoms of the patient's illness. Neurologic sequelae have included: inertia, masked facies, convulsions, hemiparesis, aphasia, and ataxia. Permanent change with respect to the above symptoms is less common than temporary change.

Most of the cases subjected to this operation were diagnosed affective reaction types (38 cases) or tension states (19 cases). High degree of persistent relief is reported for the symptoms: nervous tension, worry, anxiety, insomnia, suspiciousness, apprehension, agitation; for crying spells, suicidal ideas, depersonalization, frigidity, irritability; for introversion, indecisiveness, negativism, delusions, ideas of influence or reference. Therapeutic results reported of the affective reaction type group (38 cases): 26 patients, good; 6, fair; 4, poor; 1, dead; and 1, unevaluated. Of the 19 tension state cases: 14, good results; 1, fair; 2, poor; 2, dead. Of the 12 patients with schizophrenia there were: 5, good; 4, fair; 2, poor results, and 1, unevaluated. A few cases of psychoneuroses (anxiety state or hysteria), alcoholism, and of unclassified psychoses were operated upon with good results reported for the psychoneuroses.

The book makes interesting reading and the theories and speculations it presents are highly stimulating. Most plausible is the general concept that the thalamus represents the emotions and the cortex the ideas and plans. Cutting the connections of the cortex and thalamus thus separates worry from thinking, but unfortunately also separates drive from planning, concern with good manners from social behavior.

Two important questions are left unanswered. First, just what anatomic structures are destroyed in the operation? The fact that the part of the brain cut is not seen by the operator and the fortunately small number of deaths account for this. Serial sections of the brain at a later date will clarify this point. Second, is the operation of benefit to patients? The evaluation of therapeutic results in psychiatric disease is a difficult one. A systematic presentation of protocols of *all* cases operated would have been valuable in assisting the reader in evaluating the therapeutic possibilities of the operation. Space for this presentation might have been obtained by judicious omission of some of the theorizing about the causes of the illnesses, since the illnesses described are all diseases of unknown etiology. Whether or not the disability occasioned by the operation will in time outweigh the loss of symptomatology, the future will decide. Whether the operation or the passage of time occasioned improvement is also a major problem to be answered. Even without any treatment, the prognosis in patients with involutional melancholia featured by much anxiety and tension is good, according to some observers. If this be true, the problem of therapeutic evaluation here is exceedingly difficult.

On the whole the book is well worth reading by those interested in psychiatry, cerebral function, and experimental surgery on the human brain.

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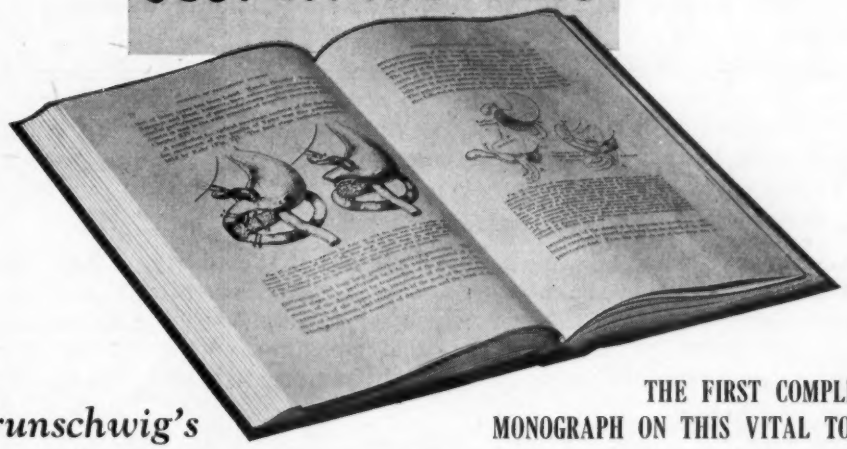

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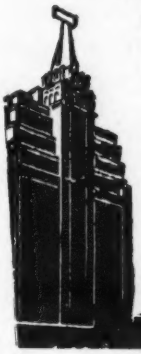

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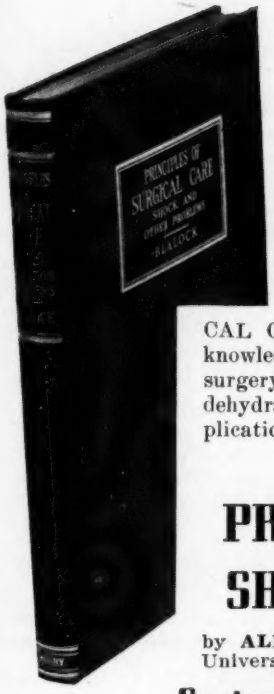

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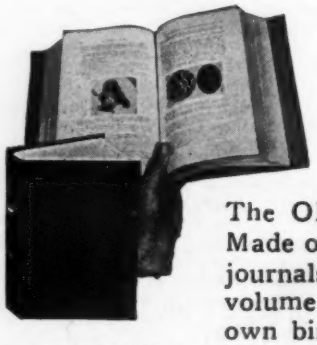

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